IN THE CHANCERY COURT OF JACKSON COUNTY, MISSISSIPPI

In re:	
MIKE MOORE, ATTORNEY GENERAL, ex rel, STATE OF MISSISSIPPI,	No. 94-1429
Plaintiff,	
versus	DEPOSITION OF
THE AMERICAN TOBACCO COMPANY; AMERICAN BRANDS, INC.; R.J. REYNOLDS TOBACCO COMPANY; RJR NABISCO, INC.; BATUS CORPORATION; BROWN & WILLIAMSON TOBACCO CORPORATION; PHILIP MORRIS COMPANIES, INC.; PHILIP MORRIS INCORPORATED (PHILIP MORRIS U.S.A.); LIGGETT GROUP, INC.; LIGGETT & MYERS, INC.; BROOKE GROUP, LIMITED; LOEWS CORPORATION; LORILLARD CORPORATION; THE COUNCIL FOR TOBACCO RESEARCH--U.S.A. INC. (SUCCESSOR TO TOBACCO INSTITUTE RESEARCH COMMITTEE); THE TOBACCO INSTITUTE, INC.; HILL & KNOWLTON, INC.; CORR-WILLIAMS TOBACCO COMPANY; GENERIC PRODUCTS CORPORATION; LAUREL CIGAR & TOBACCO COMPANY, INC.; LONG WHOLESALE, INCORPORATED; THE LEWIS BEAR COMPANY; WIGLEY AND CULP, INC. OF GULFPORT MISSISSIPPI; "A" THROUGH "Z" ENTITIES (M.R.C.P. 9(h) DEFENDANTS)	ARNOLD PLATZKER, M.D. VOLUME I WEDNESDAY, OCTOBER 30, 1996
Defendants.	

Volume I of the deposition of ARNOLD PLATZKER, M.D., taken on behalf of Defendant R.J. Reynolds Tobacco Company, at 355 South Grand Avenue, 35th Floor, Los Angeles, California 90071, commencing at 9:00 a.m., Wednesday, October 30, 1996, pursuant to Notice, before JOHANNA C. BLANKINSHIP, CSR NO. 8734.

APPEARANCES:

FOR PLAINTIFF MIKE MOORE:

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I N D E X

EXHIBITS

INFORMATION TO BE SUPPLIED

(None)

UNANSWERED QUESTIONS

(None)

LOS ANGELES, CALIFORNIA

Wednesday, October 30, 1996, 9:00 a.m.

ARNOLD PLATZKER, M.D.,
having been first duly sworn, was
examined and testified as follows:

MR. FURR: We have all introduced ourselves off the record, but let's begin by introducing ourselves and stating our affiliation.

My name is Jeff Furr, and I represent the R.J. Reynolds Tobacco Company.

MR. MINTON: I'm Mike Minton. I'm here on behalf of Lorillard Tobacco Company.

MR. PATRICK: My name is Charles Patrick. I'm with Ness, Motley, Loadholt, Richardson & Poole, and we represent Attorney General Mike Moore, the plaintiff in the lawsuit.

THE WITNESS: I'm Arnold Platzker. I'm a physician and professor of pediatrics at University of Southern California, physician and professor in pediatrics U.C.L.A. School of Medicine, and head of the division of pediatric pulmonology at U.S.C. Children's Hospital, Los Angeles, and U.C.L.A. Children's Hospital.

MR. FURR: I have one preliminary matter I want to address before we get started, and that is directed to Mr. Patrick.

In connection with the Notice of this deposition, we also served a Subpena for Production of Documents. To my knowledge, unless I somehow have not been made aware of it, no documents have been produced responsive to that Subpena; is that correct?

MR. FURR: It was my understanding there was a Subpena that was served, and my understanding is as follows; is that the court has yet to rule on what would be appropriate to be submitted in response to the Subpena. And naturally, the Subpena was not served on the witness, but we accepted service of the Subpena as the plaintiff's attorneys; that the Subpena barred certain things to which we objected and that the court has this matter under advisement and that it also asks that the documents be submitted a certain number of days in advance of the deposition.

And you are correct; that was not done. However, the doctor has brought with him -- it's my understanding -- the articles, some of the articles on which he intends to rely in his testimony in the case, as well as some notes that he has prepared to

aid him in a review or in an explanation of his opinions in this case, which he's brought with him this morning.

But to answer your question, no, we did not serve the documents as requested under the Subpena.

MR. FURR: Okay. Thank you. That's my understanding, also, with respect to the status of the issue of the documents; that they're going to be produced.

MR. PATRICK: Right.

MR. FURR: We will explore today what documents the doctor has and has not brought with him. But so that everyone's on notice now, I will state that it will our position that, rather than conclude this deposition today when we're done asking questions, we will suspend the deposition and reserve our right to continue the deposition pending review of whatever documents he has brought with him today and those that may be produced in the future.

MR. PATRICK: And pending a determination, finally, by the court on this issue.

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EXAMINATION

BY MR. FURR:

Q Dr. Platzker, have you ever been deposed before?

A Yes.

Q So are you generally aware, then, of how a deposition operates?

A Yes.

Q It's nothing more than a series of questions and answers. There are only a few things that I would ask you to try to abide by today. The first is that I will try to ask very specific questions, and I would request that you attempt to answer the questions that I ask.

And I'm sure we would all appreciate if you would not speculate if you do happen not to know the answer to a question that I ask, but instead just simply tell us that as you sit here today you can't answer the question.

If you need a break today, feel free to ask for a break at any time you want. If you don't understand a question that I ask, I'll be happy to attempt to clarify it. If you don't hear something that I ask, I'll be happy to repeat it.

Is that fair?

A Yes.

Q The first question I want to ask you is will you please describe for us your experience in treating medicaid patients in Mississippi.

A I have no experience in treating medicaid patients in the Mississippi.

Q Do you have any experience in treating patients in Mississippi, medicaid or otherwise?

A No.

Q Have you performed any studies on medicaid patients in Mississippi?

A No.

Q Have you performed any studies on Mississippi patients, whether they're on medicaid or not?

A I really can't answer that as patients that I treat in California may have come from -- some may have come from Mississippi.

Q Have you performed any studies restricted to subjects of the state of Mississippi?

A No.

Q Have you in connection with this case reviewed any studies that are restricted to Mississippi medicaid patients?

A Not that I'm aware of.

Q Have you in connection with this case reviewed any studies that are restricted to Mississippi patients?

A No, not that I'm aware of.

Q Would you describe for me your understanding of what this case is about.

MR. PATRICK: Well, let me object to the form of the question.

We provided to you the statement of what his expertise is, what his anticipated testimony will be, along with the general areas of his expertise, and the adverse effects of maternal cigarette smoking and environmental tobacco smoke, and he will be presented as an expert on those issues.

To the extent to which he understands the legal areas of this case or what this case is about I think is totally irrelevant because he is the one who is going to be asked the questions about areas of medical expertise and his expertise in medical testimony or medical issues, so that I would object to the question.

But to the extent that he can understand the question and can answer it, he can answer.

MR. FURR: Okay. Maybe you can educate me on something in this case. It is my understanding that

the last Case Management Order entered in this case was the April 21, 1995 Case Management Order.

MR. PATRICK: I don't know the date, but that may be correct.

MR. FURR: Well, it's my interpretation of that order that all objections except those that are privileged or where the form of the question are reserved --

MR. PATRICK: That is correct.

MR. FURR: -- including those involving relevance.

MR. PATRICK: That is correct.

MR. FURR: It's also my understanding that that order specifically prohibits the making of speaking objections during the course of the deposition.

MR. PATRICK: It may be. It may.

MR. FURR: I'd be happy to provide you a copy if you'd like to see it.

MR. PATRICK: Well, I don't think that will be necessary, and my objection wasn't in the nature of a speaking objection, simply to object to the intent or scope of the question that you asked. It was a form objection, and I think to the extent that it asks for totally irrelevant information, I think it is objectionable. But as I said, the doctor can answer.

MR. FURR: Fine. Fine. The objection is noted.

Q Doctor, could you explain your understanding.

A Could you repeat the question.

Q Certainly.

Could you explain to me your understanding of the nature of this case.

A I believe that the case is predicated on the hypothesis that exposure of the fetus, newborn infant, older infant child, and adolescent to the effects of cigarette smoke, whether transplacentally or in the environment following delivery or due to adolescent or preadolescent smoking has adverse effects on health and that the State has a position in that case in reclaiming the costs due to the adverse health effects of smoking.

Q Doctor, when you use the term "hypothesis," what does that term mean to you?

A Hypothesis is a theory that stands for tested.

Q Is it a theory that has not yet been proven?

A Yes. And that's why experts who might know more than the attorneys involved in the case are

asked to testify on what is fact and what is still hypothesis.

Q What familiarity do you have with the Mississippi medicaid population?

A None.

Q When is the last time that you were in Mississippi?

A I really don't remember.

Q Has it been within the past ten years?

A It may.

Q Do you know the eligibility requirements to obtain medicaid benefits in Mississippi?

A No.

Q Can you describe the demographics of the population that receive medicaid benefits in Mississippi?

A No.

Q Doctor, how did you get involved in this case?

A I was contacted by the attorneys for the State of Mississippi.

Q And who contacted you?

A Ms. Flowers.

Q Ms. Flowers?

A Uh-huh.

Q Do you know when that contact was made?

A Several months ago.

Q What were you asked to do?

A I was asked to testify on the health effects of maternal cigarette smoking and the impact of environmental smoke, cigarette smoke exposure on health of the pediatric population.

Q Subsequent to the initial contact, who else have you talked to about this case?

A Who else?

Q Let me rephrase that.

What other attorneys have you talked to about this case?

A None.

Q None other than Ms. Flowers?

A Well, except for Mr. Patrick.

Q Have you spoken with any other medical professionals about this case?

A I might.

Q Do you recall who you've spoken to?

A Not really, no.

Q Do you recall how many other medical professionals you've spoken to?

A Well, at least seven others.

Q At least seven.

Can you recall any of their names?

A Yes. They all report to me.

Q Could you provide those names for us, please.

A Yes. Dr. C. Michael Borman, Dr. Thomas Keens, Dr. Cheryl Lew, Dr. Sally Davidson Ward, Dr. Monique Margetis, Dr. Ethna MacLaughlin. I think I've covered them all.

Q And are all of these people physicians?

A Yes.

Q And they report to you in your department?

A Yes.

Q What has been the nature of your conversations with them?

A Well, first of all, to tell them that I wouldn't be available today due to the deposition and then just to mention what the nature of the deposition had to do with.

Q Have you talked with them about the issues that you are prepared to testify on?

A Not at any length, no.

Q Have you performed any original studies of maternal smoking and health effects?

A No.

Q Have you performed any original studies of environmental tobacco smoke and health?

A No.

Q What work have you done in conjunction with this case?

A Could you be more specific.

Q What work have you done to prepare for testifying in this case?

A Well, I reviewed all of the literature in my files, performed Medlines survey to determine whether there was work that I was unfamiliar with -- on the effects of tobacco smoke on the pediatric populations.

I believe that's the scope of the work.

Q Are these research files that you've maintained over time when you refer to your files?

A Over about 20 years, yes.

Q And you say you performed a Medlines search?

A Medline search, yes.

Q What topic did you perform that search on?

A On all of the respiratory and nonrespiratory health effects of tobacco exposure, smoke exposure on fetal development, the impact on

the birthing process, on the neonatal period, and on the period following infancy. I should include infancy, as well as the period after infancy.

Q As you sit here, can you approximate the number of articles that you identified with your Medlines search on these topics?

A Somewhere between 150 and 200.

Q Did you obtain copies of all those articles?

A Not all of them, no. Some were not relevant, or I didn't feel they were relevant.

Q How many articles did you obtain copies of?

A I can't really tell you the number because many of them were already in my files.

Q How many articles have you reviewed as you prepared to provide testimony in this case?

A Somewhere between 150 and 200.

Q And you reviewed the complete article for all of these?

A Not all of them, no.

Q How did you determine which articles to review and which ones not to review?

A Oh, that's fairly simple. If an article cut in the groundwork with which I was unfamiliar, I

obtained the article and read it. And if it merely replicated work with which I was already familiar, I probably might not have obtained it other than reading the abstracts.

Q And have you maintained those articles in some type of file that you reviewed?

A Oh, I have extensive reprint files so that many of them are already in reprint files.

Q Doctor, are you being compensated by the State of Mississippi for your work in this case?

A I haven't submitted any bills to them.

Q Do you intend to submit a bill to them?

A Surely if there are expenses for which I need to be compensated, I will.

Q Do you intend to submit a bill for the time that you devote to the case?

A I haven't made a decision on that, no.

Q Have you done other type of consulting work -- strike that.

Have you done consulting work for other clients?

A Other clients --

Q Other than the State of Mississippi.

A On the tobacco issue?

Q On any issue?

A Yes.

Q What is your normal hourly rate?

A It's changed over the years. It's between 250 and $350 an hour depending on whether I have to go to court or I'm just doing research work for them.

Q If you submit a bill to the State of Mississippi, what hourly rate will that bill be based on?

A Again, depending on whether I go to court, travel, et cetera, miss time at work, that kind of issue would be taken into account.

Q And what's the likely range that might be utilized?

A $250 an hour for pure research work. $350 an hour for deposition and court appearances.

Q How many hours have you devoted to this case up until today?

A Probably about 12 to 15 hours.

MR. FURR: I want to hand the documents to the court reporter and ask it be marked as Platzker Exhibit A for identification.

(Defendant's Exhibit A was marked for identification and is attached hereto.)

BY MR. FURR:

Q Dr. Platzker, you've just been handed what's been marked as Platzker Exhibit A for identification.

Are you familiar with that document?

A Yes.

Q Did you draft the first page of that document?

A I have participated in its drafting.

Q I have a few questions I want to ask you.

If you would, please, go to the middle paragraph --

A Uh-huh.

Q -- and the first full sentence, could you tell me what is meant by the last clause of that sentence that reads:

"...the short and long-term health effects of maternal smoking on newborns, infants, and children. . ."

A Yes.

Q Could you tell me what is meant.

A Yes. The "short term" meaning -- I would prefer to use the acute effects, meaning the acute effects on the fetus during labor and the acute

effects on the newborn and the infant in the neonatal period, which is the first 30 days after birth.

"Longer term" meaning through the pediatric experience, which usually ends somewhere around 18 years of age.

Q Without telling me what your opinions are at this point, would you tell me the effects that you are identifying as the acute effects?

A The acute effects entail any compromise of the health of the fetus, the impact of those health effects on the process of labor and delivery, and on the newborn infant.

Q What do you mean by "long-term effects"?

A Long-term effects are the impact of the exposure to maternal cigarette smoking either in the uterus during embryonic and fetal life, on the maternal smoking after birth, and actually, the result of the exposure to smoking either as an environmental hazard or as a hazard from the child or adolescent smoking, as well.

Q Without going into the bases for all of your opinions at this time, can you give me a list of the long-term effects?

A I don't know. I could share an outline of that with you.

Is that --

MR. PATRICK: Sure.

THE WITNESS: In this list I've broken down the effects --

BY MR. FURR:

Q Doctor, could we mark it for identification first so we can all keep track of what this means later.

(Defendant's Exhibit B was marked for identification and is attached hereto.)

BY MR. FURR:

Q Doctor, the list that you are now referring to has been marked for identification as Platzker Exhibit B for identification.

A Right. I've identified here the fetal tobacco syndrome and given some references to the criteria for this, then the acute findings, and those findings that are more long term, which may have something to do with fetal exposure, but also the continued exposure to side-stream or environmental tobacco smoke inhalation.

And I added another paragraph on my analysis of the -- organizing the data into some rational thought on what the potential deleterious

effects are.

MR. FURR: Take a break for one minute.

(Discussion held off the record.)

BY MR. FURR:

Q Doctor, would you describe for us, please, your current position and responsibilities in your position.

A Yes. I'm the head of the division of pediatric pulmonology at University of Southern California School of Medicine at Children's Hospital Los Angeles, and Division of Pediatric Pulmonary Medicine at the U.C.L.A. School of Medicine Children's Hospital.

Q Do you currently see patients?

A Yes.

Q Do you see patients on public assistance?

A Yes.

Q What areas do you consider yourself expert in?

A On the -- I think as I pointed out in my curriculum vitae, where it could be summarized as the childhood antecedents of adult lung disorders.

Q What does that mean?

A Well, everything from the impact of

neonatal respiratory disease as it might affect the older child and adult, and that would be both acquired and congenital disorders of the lung; congenital being something like an inherited disorder such as cystic fibrosis or perhaps respiratory distress syndrome of the newborn, or evidence of impaired fetal homeostasis, such a meconium aspiration syndrome or pulmonary hypertension of the newborn infant, congenital diaphragmatic hernia, all of which impair respiratory function in the newborn period but have an impact on later development of the lung and lung function.

Q Doctor, in your 26(B)(4) Expert Statement, which is marked as Exhibit A, in the last sentence it is stated that you may also offer opinions regarding the expense of these diseases and treatments?

A Yes.

Q Are you, in fact, prepared to discuss the expense of these diseases and treatments?

A To some extent, yes.

Q Are you prepared to discuss expenditures by the State of Mississippi --

A No.

Q -- related to these treatments for

medicaid patients?

A No. I have no experience in Mississippi.

Q Finally, Doctor, on your 26(B)(4) Statement, the last sentence on the first page states that, as a basis of your opinion, you include your review of the information, testimony, and documents concerning this case.

Other than the literature review that you've already described for us, are there other documents that you base your testimony on?

A No.

Q Doctor, you're not an obstetrician, are you?

A No.

Q Do you consider yourself expert in obstetric issues?

A No.

Q You're not on epidemiologist?

A No.

Q You're not a toxicologist?

A No.

Q You're not a biostatistician?

A No.

Q You are not a statistician?

A No.

Q Do you base your opinions in this case in whole or in part on epidemiological data?

A Yes.

Q Do you consider yourself expert in interpreting epidemiologic data?

A No.

Q Can you describe the nature of an epidemiologic study?

A Can I describe -- with respect to what?

Q The field of epidemiology in general. What type of inquiries are made? What is the nature of an epidemiologic study?

A In any area it would be to assess how populations, specific populations are affected by certain exposures or habits or the impact of various stimuli on particular populations.

Q So you would agree that epidemiology is the study of populations as opposed to individuals?

A Yes.

Q Is epidemiology largely a statistical study?

A To a great extent.

Q And what is the product of an epidemiologic study?

A Could you be more specific.

Q Yes.

Aren't the calculation of relative risk or odds ratios the product of epidemiologic study?

A Yes.

Q When you learn of an epidemiologic study reporting a finding that is of interest to you, do you take that study on face value, or do you review the study for yourself?

A I, in many respects, have reviewed epidemiologic studies and reviewed the data from the studies.

Q What criteria do you apply in evaluating the quality of an epidemiologic study?

A First of all, on whether the study has been accepted by an important peer review journal. I think that's first and foremost.

Q What other criteria do you use?

A I don't think -- I think on the basis of the scientific data and whether it is testable and whether the hypothesis has been tested rigorously.

Q When say "on the basis of the scientific data," what do you mean?

A Well, whether the -- whether it is important scientifically, that is, not just counting

grains of sand but relating the counting of the grains of sand to some scientific principle which is really being tested.

Q When you say that one of the criteria that you apply is whether the hypothesis is testable, what does that mean?

A Well, there are an accepted body of statistical analyses that can be placed on any scientifically accumulated data that -- for which there are -- a degree of certainty can be assessed to it, a "P" value, or another regression analysis, something where we can look at whether populations fall within standard deviations of a particular mean or whether they are outside of them.

Q So am I correct that you attempted to determine whether acceptable statistical tests have been applied to the data?

A Yes.

Q What type of epidemiologic study design do you consider to be the most reliable?

A That's a very broad question. I really can't answer that other than it's so broad that it's impossible to answer your question.

Q Let me ask it this way.

Among the study designs of randomized

double blind crossover, number one; and number two, prospective or cohort study designs; and number three, retrospective or case control study designs, which do you consider to be the most reliable?

A Well, I think it depends on the nature of the question being tested. For some randomized control crossover studies are appropriate; for others some form of case control and looking at specific populations; and then third, retrospective studies are probably in any area the weakest of the studies.

Q And, in fact, Doctor, aren't case control studies frequently referred to as "retrospective studies"?

A They can be, yes.

Q Why do you consider them to be the weakest of studies?

A Well, retrospectively, when you look at records, the data is only as good as data recorded. And having been involved many times in looking at retrospective data, we find that it isn't collected in a complete enough fashion to really test or answer the questions.

Q Other than incomplete collection of data, are there other types of flaws that case control or retrospective studies are especially prone

to compared to the other study designs?

A Well, I mean, there are many problems with retrospective studies, everything from the idea that it looks at past experience and is not always relevant to present experience to the difficulties in analyzing any question where the data hasn't been collected in an orderly fashion to perhaps -- and/or complete enough to answer the present question under consideration.

Q Are there any other types of problems with retrospective studies?

A I'm sure there are many that statisticians would point out, but for me, those are the basic ones.

Q Doctor, are you familiar with the term "recall bias"?

A No, I'm not.

Q Doctor, when you review a topic and find that among the epidemiologic studies of that topic you have some studies which report an association and some which do not, how do you go about harmonizing those results?

A How do I go about it?

Q Yes.

A Actually, I find that one of the more

difficult issues that anybody has in analyzing the data. Certainly, there's more recent methodology for looking at it, and that would be something like a meta-analysis where similar studies are analyzed collectively to give you a larger number of cases to review and where the studies -- certainly, I'm not an expert on meta-analysis -- but where the data is found by analysts, biostatisticians, epidemiologists to be compatible enough so that a larger end can be taken into account.

The larger the number, the lower the degree of variability, perhaps, and you can apply stronger statistical analyses to it.

Q Doctor, did you perform a meta-analysis of the studies of any of the topics that you're prepared to provide in this case?

A No.

Q Have you personally ever performed a meta-analysis?

A No, I haven't.

Q When you said that one must assess whether the data are compatible enough to combine a meta-analysis, what do you mean?

A I think that the bodies of information have to be comparable enough so that -- such that

variables such as race, socioeconomic status, nutrition, et cetera, can be taken into account. And those variables can be studied independently.

Q Is it your opinion, then, that noncomparable studies should not be combined in a meta-analysis?

A Well, I'm not qualified to make that statement.

Q What got us into all this was I asked you the question of how you go about harmonizing inconsistent epidemiologic studies.

And other than through some type of formal approach, like a meta-analysis, how else do you personally go about harmonizing inconsistent studies?

A Inconsistent studies? Could you explain that?

Q The predicate that I used initially was, if you perform a medline search, for instance, on a topic, and you retrieve ten studies, ten epidemiologic studies, if five of them find an association and five do not, how would you attempt to harmonize those results?

A Well, that's a very good question. I think the first thing would be I would have to look

at the studies and the populations studied and determine whether any of these factors that I've mentioned have brought any bias into the study population or whether perhaps the effect is restricted to a particular population.

Q In fact, Doctor, isn't it the case that the odds ratio relative risk produced by an epidemiological study applies only to the population that was under study?

A I think that's true of any study, that is that the study is only as good as the population and its homogeneity.

Q And isn't it also the case that one should not attempt to apply the relative risk for a factor observed in one population to attempt to project the risk in another population that has different characteristics?

A To some extent I agree with you.

Q Could you explain first the extent that you agree -- the extent to which you agree with me.

A I think if -- unless you have a fairly complete understanding of the population being studied, you cannot determine whether that population and the results obtained in a study of that population can be applied to another one.

Q To what extent do you disagree with me?

A That's the extent of my understanding of the issue.

Q Okay. We'll come back to this.

A Let me clarify. For example, what I would suspect would be a problem in a particular study that one might assume that you couldn't look at Filipinos in Manila and extrapolate a result from the study of Filipinos in Manila to a similar Filipino population in Los Angeles, San Diego, or New York City.

Now, on the other hand, if we knew enough about those populations and the socioeconomic level, the nutritional level, whatever, they may be comparable, and you may be able to look at the results of the study's cross-geographical areas.

Q Is it your position, then, that you should only extrapolate the results from one study to another population if you can determine that the populations are comparable on the factors that you believe important?

A Exactly.

Q Thank you.

Let's talk about a very basic epidemiologic principle, and that is, as we've

discussed, the product of an epidemiologic study is an odds ratio or a relative risk depending upon the nature of study.

And isn't it true that what those numbers represent are statistical correlations?

A Yes.

Q And statistical correlation does not necessarily mean that there is a causal relationship; isn't that true?

A I don't agree with that.

Q Surely it is not your position that all statistical correlations reflect causal relationships?

A It depends on how narrow or how broad the statistical analysis is. Have we narrowed the statistical possibilities?

Let's look at a particular hypothetical case; color of hair. And we're looking at cross-populations. If the color of hair is -- if we then extrapolate the color of hair cross-populations, well, it's hard to say that we've controlled for the many variables.

But if all the variables have been controlled for, then an odds ratio -- if we look at factor A versus factor B, the odds ratio, if we've

controlled for the dependent variables, we may really be able to look at cause and effect.

Q But I take it that the key is whether you've controlled for the dependent variables?

A Definitely.

Q So when confronted with an odds ratio or relative risk, one cannot reach causal conclusions until one assesses whether the dependent variables have been controlled for?

A Exactly. I'd agree with that.

Q Doctor, earlier you mentioned -- in response to one of my questions, you mentioned the concept of "P value."

Can you explain what you meant by "P value"?

A P value is the statistical issue that has to do with the amount of certainty that the hypothesis has been tested.

Q Are P values one of the tests that are used to assess the role of chance --

A Yes.

Q -- random variation in producing the observed tests?

A Yes.

Q Are those frequently referred to as

"tests of statistical significance"?

A Yes.

Q What is the importance of testing for statistical significance?

A The importance is to try and validate a hypothesis or find that the hypothesis is unsupported in the data.

Q If a study reports an elevated result that is not statistically significant, what does that mean to you?

A Using the P value alone doesn't mean very much.

Q The study does not mean very much?

A No. The P value, unless I've looked at the other -- how the P value is obtained, the population studied, the number in the population, whether there are variables that have been controlled for, the P value is irrelevant.

In other words, if classical statistical guidelines have been followed, then the P value is helpful in analyzing a study.

Q And is a P value of .05 the normal standard that is demanded of scientists in assessing the statistical significance of a study?

A Usually less than .05.

Q Unless the P value is less than .05, the study is typically considered not to have produced a statistically significant result?

A Yes. That, for most journals, would be considered a nonstatistically significant piece of data.

Q And isn't the problem with statistically nonsignificant data that we don't have the normally required confidence that we want that random variation has not been ruled out as producing the observed risk?

A Yes. That would be one of the analyses that could be proposed.

Q Are you familiar with the epidemiologic issue that is sometimes referred to as "statistical multiple comparisons"?

A I'm not an expert on that, no.

Q So you're unfamiliar with that term?

A Not unfamiliar with it. I'm not well enough acquainted with it to discuss it.

Q You can't discuss it today?

Let's approach this statistical significance P value issue from a little different perspective.

Isn't it also true that the only thing

that is measured by the P value or other test of statistical significance in the random variation factor -- let me rephrase.

When you evaluate the statistical significance of a result, isn't it true that all you are evaluating is the confidence that one can have with respect to random variation that's produced the results?

A Yes. That's my interpretation.

Q Tests of statistical significance do not tell you anything about the role of dependent variables in producing the results; right?

A I would remind you that I've already commented on that, that the dependent variables can be taken into account such that the P value is obtained on a homogeneous enough population that it does bear some reliability.

Q But merely scrutinizing the P value alone, does that answer that question for you?

A That's the point I made before.

Q Let's talk about another epidemiologic concept. I want to talk to you about the issue surrounding the interpretation of weak associations.

I'd first like for you to state your views on the -- in general, on the utility or

reliability of epidemiologic associations of 2.0 or less.

A Are you talking about odds ratio of 2.0?

Q Yes, sir.

A And what, specifically, did you want me to comment on?

Q I'd like for you to comment on the reliability of associations or odds ratios in that range compared to larger odds ratios.

A Well, the higher the odds ratio, the more the risk is of the particular thing occurring in an assessed population.

Q Isn't it also true that the lower the odds ratio, the more difficult it is to control for these dependent variables?

A Well, that would be something you'd want to ask a statistician because, of course, again, the number in the particular population bears some scrutiny; that is, if the odds ratio is on ten subjects and the number, the odds ratio is less than two, you would be concerned.

But if we're talking about ten million in that population, less than two odds ratio with the dependent variables have been taken into account really bears some concern and scrutiny.

Q Are the dependent variables more difficult to take into account in odds ratios less than two?

A Well, then, as I said, I think you'd have to ask for a statistician to give you the pros and cons of that.

Q That would be something outside your area of expertise?

A Exactly.

Q I take it that you have not kept up with the epidemiologic literature that is evolving with respect to the interpretation of odds ratios and relative risk of 2.0 or less?

MR. PATRICK: Objection to form.

You can answer.

BY MR. FURR:

Q You can answer, Doctor.

A I'm not an expert in that area.

MR. FURR: Let me mark something here. Let's mark this as Platzker Exhibit C for identification, please.

(Defendant's Exhibit C was marked for identification and is attached hereto.)

///

BY MR. FURR:

Q You've just been handed an article entitled "Epidemiologic Faces Its Limits." It's been marked as Exhibit C for identification.

I'd like for you to take a look at the highlighted material on Page 164. You're of course welcome to look at anything else you want to in the article.

A I haven't read this all, but what issue --

Q I think the questions that I will ask you will not require you to read it all, I don't believe.

A Okay.

Q I first want to ask you about the language that is highlighted on the exhibit in the middle column on Page 164. And that is comprised of a quote by Michael Thun that states:

"With epidemiology you can tell a little thing from a big thing. What's very hard to do is to tell a little thing from nothing at all."

And my simple question is, do you agree with that statement, Doctor?

A Well, I think in general principle, I

would have to agree with it.

Q Thank you.

I now would ask you to turn to Page 168, and I hope you also find some highlighted language there in the first column.

Do you have it there, Doctor?

A Yes. What would you like?

Q I'm going to ask you a question of the same nature. The highlighted language there is a quotation from Alex Walker, an epidemiologist at Harvard. The quote reads:

"I have trouble imagining a system involving a human habit over a prolonged period of time that could give you reliable estimates of risk increases that are of the order of tens of percent."

And my question is, do you similarly agree with that statement?

A I think this statement looks like it was taken out of context, so I'm not really -- it would have to be more specific than a statement to -- for me to comment on. As I've pointed out, I'm not an epidemiologist or statistician, so I would probably need more clarification of what is meant by this.

Q So you might agree with it in the context of certain types of studies but not in the context of other types of studies?

A Exactly.

Q You can't agree or disagree with it as a general proposition?

A Right.

Q Fair enough.

Doctor, this morning we've been using the terminology of controlling for dependent variables.

A Yes.

Q Does that terminology -- is that terminology synonomous with the concept of controlling for confounding?

A Not really.

Q What are the differences?

A Well, dependent variables are variables that are totally related, and confounding variables are other variables that may impact on the analysis of the data but may not be as immediately important.

That's my understanding. It may differ from classical statistical thinking.

Q You're not sure as to whether it differs or not?

A Well, I'm just giving you my opinion.

Q What is your understanding of the concept of controlling for confounding in epidemiologic studies?

A I think you're asking for an expert analysis, and as I've pointed out, statistical analysis is not my --

Q Okay.

You've stated that, in part, your opinions are based on epidemiologic studies.

A Yes.

Q And you have told us that you don't take the studies merely at face value, but that you do attempt to scrutinize the study --

A Exactly.

Q -- to determine the scientific validity of the study.

When you do so, do you attempt to determine whether or not confounding has properly been controlled for?

A I look at the study and determine specifically whether there is a strong hypothesis; second of all, whether the hypothesis has, in terms of methodology, been well-tested; third, whether the important variables have been taken into account.

And more importantly, when I'm concerned about the quality of the study, I consult statisticians who can confirm or reject whether the study has been well-constructed.

Q Doctor, when you say you look to determine whether the important variables have been taken into account --

A Yeah.

Q -- is that an exercise in assessing whether confounding has been controlled for?

A All variables.

Q So that would include the assessment of confounding?

A Yes.

Q How do you determine whether confounding has been controlled for in a study?

A I think I've gone over this already with you.

Q I'm sorry. I missed it if you did.

A Well, as I said, I pointed out that I look at all the variables that have been listed. And if you wanted to me to be more specific, I think I have.

Q Can you state for us in general terms what types of variables should be controlled for in a

study?

A I think we've covered that before, but if you'd like, I think that, first of all, to look at the population, determine whether it's a single population or whether what is being studied are multiple populations; if it's a single population, whether age, sex, race, socioeconomic variables have been taken into account, whether the population is affected by various genetic, metabolic, environmental phenomena that are different.

Q Should one attempt to determine whether other known risk factors for the disease that you're investigating have been taken into account?

A I think that's realistic.

Q Among the lists that you gave us with socioeconomic status, is it true that socioeconomic status is a very important variable to take into account?

A It is one of many variables.

Q Do you consider it to be an important variable?

A It's an important variable depending on the study to be performed.

Q How does socioeconomic status work as a risk factor for disease?

A Well, it can be as vague as where the person lives, the number of people in the room, in the domicile, the closeness or proximity to environmental hazards, nutrition, the ability of the -- in pediatrics of the family to care for the child. Those are some of the possible impacts of socioeconomic status. And education may play a role in that, too, but. . .

Q Doctor, in response to an earlier question, you indicated that -- if you have concerns about the approach that was taken in a study to control for other variables, that you sometimes consult a statistician to help you assess the approach; is that correct?

A Yes.

Q Have you consulted a statistician in conjunction with your review of any of the literature that you reviewed to prepare your opinions in this case?

A Not specifically for this deposition, no.

Q Have you consulted a statistician at all?

A I'm involved in a study that we've compared our data and -- a study that had nothing to

do with tobacco smoke with a researcher who is dealing intimately in it and, adequately to do a study, we needed to carefully analyze the statistical data of the other investigator so. . .

Q That that was not done in conjunction with this case?

A No.

MR. FURR: Can you mark this as D, please.

(Defendant's Exhibit D was marked for identification and is attached hereto.)

BY MR. FURR:

Q Doctor, before we look at what's been handed to you marked as Platzker's Exhibit D for identification, I want to ask you, do you read the "New England Journal of Medicine"?

A Pardon me?

Q Do you read the "New England Journal of Medicine"?

A Yes.

Q It is one of the premier medical journals in this country and in the world, isn't it?

A Yes.

Q In fact, did I see in your curriculum vitae that you are a reviewer for the journal?

A Yes.

Q Do you know Marsha Angell?

A Not personally.

Q Do you know her by reputation?

A By when she sends me an article to review. That's about the extent of.

Q So you receive articles from Dr. Angell?

A Yes.

Q And that's in conjunction with your performance of reviews for the journal?

A Yes.

Q Do you know what Dr. Angell's position is at the "New England Journal"?

A I think she is one of the two editors. She and Dr. Kassirer are the two editors. She's either managing editor or executive editor, one of the two.

Q Is that a fairly esteemed position?

A I can't say how esteemed it is. It's an important position within the journal.

Q It's an important position?

Could you take a moment and look at that article, please. The questions that I'm going to ask you will be focused on the highlighted material in the article.

(Discussion held off the record.)

(Recess from 10:10 a.m. to 10:15 a.m.)

BY MR. FURR:

Q Doctor, let me begin by apologizing because I lied. I'm going to ask you a question or two that's not highlighted. We'll try to direct you to the language.

A Okay.

Q Have you had an opportunity to at least take a look at the exhibit now?

A Yes.

Q Let me ask you about the very first sentence. It reads:

"Anyone who follows the medical literature knows that 'socioeconomic status' is a powerful determinant of health."

Do you agree with that, Doctor?

A Yes.

Q Let's go to the bottom of the first column on Platzker's Exhibit D, and I want to ask you about the first sentence in the bottom paragraph. And that reads:

"So closely does socioeconomic status correlate with health that it confounds

the interpretation of much clinical research."

Do you agree with that, Doctor?

A I think it's probably true.

Q The next sentence reads:

"For example, studies of the effect of passive smoking on childhood asthma are uninterpretable unless an attempt is made to control for socioeconomic status."

Do you agree with that, Doctor?

A I don't know.

Q You do not agree with it?

A Not entirely without qualification.

Q Could you explain, please.

A Yes. It's sort of interesting. I'm involved in a study looking at asthma, and it's across socioeconomic groups. And we find that a similar percent of children regardless of socioeconomic groups have asthma.

Now, I would agree that socioeconomic status has a great impact because the poor -- what we've looked at is the presence of an illness but not the extent of an illness, and that is it may be terribly dependent on the healthcare the child gets,

the nutrition the child gets as to the expression of a problem.

Q Isn't the question that we're looking at here, Doctor, not whether children in different socioeconomic groups might have a similar incidence of the disease, but whether those children have the disease for similar reasons?

Aren't those separate questions?

A Oh, yes.

Q Let's look at the next sentence on the bottom of the Page 126, the first column:

"Without such control, it is impossible to know whether the increased prevalence of asthma in the children of smokers is really because of passive smoking or because smokers are more likely to be poor and poverty itself is associated with a higher prevalence of asthma."

Do you agree with that?

A No.

Q Why is that?

A Because I don't think there is clear, irrefutable data that children of smokers have a higher incidence of asthma.

Q So you would agree that the evidence

does not demonstrate that children of smokers have a higher incidence of asthma?

A Asthma being a very specific disease, I didn't say they didn't wheeze, but I'm questioning whether the data is strong enough to say that they have more asthma.

Q Okay. Thank you.

A I'm saying that I don't agree with the statement as written.

Q I understand. I appreciate your explanation.

Let's go to the next column, the highlighted material, which reads:

"Indeed, if the direct effect of a variable under study -- for example, passive smoking or exposure to lead -- is small, and the effect of socioeconomic status is large, it may be difficult to correct for socioeconomic status adequately."

Do you agree with the principle espoused in that sentence, Doctor?

A I think the two may be related.

Q I'm sorry. I don't understand.

A I think the argument that Dr. Angell is

taking in this position she then modifies as she goes on with her explication. Let me use lead. If -- because it's not something we're discussing today. I think the two are related, that is, that we showed that children within 500 yards, living within 500 yards of a freeway during the era where lead was in gasoline had higher lead levels.

There the two were related, that is, that the exposure, if they lived a mile away, would be much less. But the lowest-cost housing was closest to the freeway so that the two were interrelated. And if they were less poor, they would have been less exposed.

Q And so --

A So in this instance, the proximity to the freeway was as important a variable as their socioeconomic status.

Q When you have such a situation, how do you sort out which variable is contributing to the incidence of disease?

A Well, the studies that were done on lead showed that the children who lived within 500 yards of the freeway or a hundred yards -- I can't remember whether it was 1- or 500 yards -- had a higher lead level than children living farther away.

Secondarily, an analysis of the socioeconomic status and understanding that the areas closest to freeways were least desirable in terms of noise, smoke, whatever, that led the researchers in this instance to determine that it was the proximity to the freeway, as well as the socioeconomic status that were responsible and that lead was -- and its exposure, the exposure of the child to lead was one of the -- really the most important issue.

Q Let me ask you one final question on Exhibit D, and that is the first sentence of the next paragraph, which reads:

"Yet, despite the importance of socioeconomic status to health, no one knows quite how it operates."

Would you agree with that principle, Doctor?

A Again, I think this is a summary of many separate thoughts, and I would agree with it from the perspective that socioeconomic status has many, many issues within it, some of which we've already discussed. And to dissect one of the issues from another is very, very difficult.

Q Very difficult.

Because socioeconomic status may be a

proxy for many variables?

A Exactly.

Q Doctor, as we complete the examination of the exhibits, make sure we provide them to the court reporter, please. Thanks.

Doctor, isn't another problem in the interpretation of epidemiologic studies that one can only adjust for the dependent variables or confounders that one knows about?

A I think that's a non sequitur. It's true.

Q In fact, if you do not know yet that a certain factor may subsequently be found to be a risk factor for disease, then you don't know the control for it in a study that you're currently conducting?

A Yes.

Q Would you agree, Doctor, that for all of the diseases or medical conditions that you have described in relation to your 26(B)(4) Statement and your opinions in this case that we do not yet know all of the causes for those diseases?

A All of the causes --

Q For any of the disease that you're prepared to discuss in this case.

A I would agree that there is knowledge

yet to be determined.

Q And it will not surprise you, will it, if then years from now we knew of new causes for any of those diseases?

A Possibly.

Q Doctor, are you familiar with the concept of publication bias?

A Publication bias?

Q Publication bias.

A No, I'm not. Perhaps if you could explain the term from your perspective. . .

Q If you're not familiar with that term, we'll just move on.

Q I want to talk about another statistical issue.

Are you familiar with the term "attributable" --

(Interruption.)

BY MR. FURR:

Q Do you have a page that you need to respond to?

A Yeah.

MR. FURR: Let's take a break.

(Brief recess.)

(Mr. Minton left the proceedings.)

BY MR. FURR:

Q Doctor, do you have an opinion as to whether journals preferentially publish studies that tend to report a risk as opposed to studies that found no risk?

A That's a good question. I would think it would vary with the journal.

Q Might it also vary with the topic that's being examined?

A Well, I would think it would be more related, and this is opinion rather than fact. I think it would be more related to the journal and its publication policies.

Q Doctor, do you have an opinion as to whether in epidemiologic studies of the case control nature, whether cases tend to preferentially recall exposures as opposed to controls?

A Could you repeat that.

Q Yes.

This really gets back to a question I asked you earlier. I used the terminology "recall bias," and the concept that I'm getting at is I'd like to know your opinion on whether in case control studies for whatever combination of motivations that might motivate the participants that cases

preferentially tend to recall their exposures compared to controls.

(Mr. Minton entered the proceedings.)

THE WITNESS: That's a good question. I haven't thought about that before, but I would -- I would think that it would depend upon the questionnaire.

BY MR. FURR:

Q In your review of the literature in preparation for expressing your opinions in this case, I take it, then, that you made no effort to assess recall bias in studies?

A As I said, I think that would depend upon how careful the investigators were to be unbiased towards the question they were studying.

Q In connection with your review, did you obtain the questionnaires for any of the studies that you reviewed?

A No.

Q Doctor, are you familiar with another statistical term, and that is "population attributable risk" or "attributable risk"?

A Well, I'm not -- I've heard the term, but I'm not totally knowledgeable about it.

Q Are you familiar with efforts to, on a

statistical basis, attribute a certain percentage of disease in a population to a given -- to a specific factor?

A Uh-huh.

Q And what's your familiarity with that process?

A Well, I think, as with any other type of research, it would be important first to identify what risk you're looking at and the specific variables that might interact with that risk.

Q In connection with this case, have you made any effort to attribute a percentage of disease in the Mississippi medicaid population to either maternal smoking or environment tobacco smoke exposure?

A As I said before, I really haven't reviewed any data from Mississippi --

Q So --

A -- specifically.

Q -- the answer to my question is "no." Thank you.

Dr. Platzker, what's your understanding of the concept of dose response in an epidemiologic study?

A Well, that's a pretty general question,

and I'm not certain what -- how you'd like me to answer it.

Q Is it important to assess dose response in epidemiologic study?

A Yes.

Q Why is that?

A Well, there are a number of potential harmful agents that, when taken in below the threshold of the body to deal with them, may not cause injury so that the specific exposure and the concentration of whatever the agent is within the body -- the body's ability to handle it becomes important in virtually all the studies.

Q If within the epidemiologic data acquired in a study one sees no evidence of increasing risk for increasing exposure, what does that mean?

A Well, it means either that the particular population under study has greater capacity for handling the various -- the specific agent under study or that the agent under study has a wide range of safety such that in the doses studied the population will not respond adversely to it.

Q Would you agree that it is a fundamental medical principle that dosage is critical in

assessing the effects of an exposure?

A No, not entirely. I would agree with the importance that dose is very significant, but I think it's the organism's ability to handle any specific dose.

For example, there are certain populations that are incredibly capable of handling almost any dose of a particular agent because they can metabolize it and detoxify it; whereas other populations -- for example if you have liver disease and you're talking about certain drugs, then you wouldn't want to use them in that population because they are detoxified in the liver, and even a small dose may cause deleterious effects on the health.

So it's a combination of the dose and the ability to handle the particular agent in question.

Q Earlier you used the term "threshold."

What did you mean by "threshold"?

A Threshold is the level at which any product either in the blood or perhaps the tissue causes the effect, an effect on body metabolism.

Q I'm sorry. Could you repeat that. I didn't quite hear it.

A In other words, given a therapeutic

agent, the threshold for that agent is the level either in the blood or the tissues for which that agent is effective.

I'll give you an example. If a patient has seizures and you give the patient phenobartital, the level of phenobarbital in the blood can be extrapolated to whether appropriate seizure control may be obtained.

Q Does this concept of threshold also extend to toxic exposures as opposed to therapeutic exposures?

A It can.

Q For all of the diseases or maladies that you identified in your 26(B)(4) Statement, would you agree that there is a threshold of either maternal smoking or environmental tobacco smoke exposure, as the case may be, below which no observable increase in risk is found?

A Again, it would depend on the population, that is, the ability of the population to handle the amount of cigarette smoke --

Q Well --

A -- or whatever agents in the cigarette smoke.

Let's arbitrarily say that there's a

particular population that can't metabolize nicotine so at a small dose of cigarette smoke, that population may experience a higher level than another population who would not be affected at all.

Q I want to reask that question. I want to ask it in the context of an epidemiologic approach to assessing risk in populations.

And the question is, wouldn't you agree that there is a threshold of exposure for either maternal active smoking or environmental tobacco smoke exposure below which no risk has been observed in the epidemiologic studies for all of those diseases you've identified in your 26(B)(4) Statement?

A Again, I'd have to say that much of this is dependent on the ability of the particular group to be able to handle the agents in smoke, that is, to detoxify them and to eliminate them.

Q Can you point to any epidemiologic study that provides evidence of the absence of a threshold for maternal smoking or environmental tobacco smoke exposure?

A Could you repeat that.

Q Yes.

In your review of the literature, did

you come across any epidemiologic studies that showed an observable increase in risk all the way down to the lowest exposure studied?

A Well, the data is quite variable in terms of what constitutes the lowest exposure; that is, there are some studies that go -- that studies groups with no smoke exposure, 1 to 9 cigarettes a day, 10 to 20, 21 or more, so that they are variable studies. And I think the impact in all the studies is typical of a dose-response curve. That is, at the lower exposure levels, there's a less profound effect.

Q Doctor --

A It's highly variable.

Q It's highly variable?

A Uh-huh.

Q And didn't you come across studies that showed no evidence of a dose-response effect?

MR. PATRICK: Objection to form.

You can answer.

THE WITNESS: No, I don't believe I did. I'd have to review -- as you can see, I've read a lot of different articles, and I'd have to review whether there were some that didn't show a dose response.

///

BY MR. FURR:

Q If one wanted to know the risk for environmental tobacco smoke exposure or for maternal smoking for a specific disease or malady in the Mississippi medicaid population, wouldn't the best possible evidence come from a study of the Mississippi medicaid population?

MR. PATRICK: Objection.

You can answer.

THE WITNESS: I don't think so. It's a much more complicated issue than just whether they live in Mississippi or not.

BY MR. FURR:

Q How would one go about examining those -- what's the best possible way to examine that question?

A Maybe you want to restate the question.

Q Certainly.

If I want to know the risk of environmental tobacco smoke among -- for a disease in children under 19 years old that were covered by medicaid in Mississippi, how should I go about studying that question?

A First of all, I'd want to identify what risk factors you're looking for. I mean, what impact

of smoking are you looking at?

Second of all, in what part of childhood are you looking at? Is this the influence during pregnancy? I mean, you could start farther back and look at the influence on fertility or on the fetus; on the embryo; fetus, newborn, There are areas in development that you might want to look at, so that just looking from conception through childhood, it would be very, very difficult to just do that broad a study. There would be too many variables.

Q Those are excellent points, and my questions was overbroad.

If we do focus it in the way that you're suggesting now, would I be best served by looking at that group within the Mississippi medicaid population, or should I look elsewhere?

A Well, to be honest, if research has been done and published that already answers the question relative to the risk for various phenomenon, the Mississippi populations needn't be studied if other populations have been adequately studied and are analogous to the Mississippi population.

Q If they are analogous to Mississippi population?

A That's right.

Q So that's the judgment that would have to be made?

A Yes.

Q Let's switch gears here, get away from this epidemiologic, to some extent, anyway.

A Uh-huh.

Q Is preventive healthcare important in preventing disease in children under the age of 19?

A It's very important.

Q Why is that?

A Well, if you can avoid exposure of a developing organism to a particular threat to health status, you've avoided not only the acute illness but the potential sequelae of the illness.

Q Do you have an opinion as to whether proper utilization of preventive healthcare also decreases the cost of medical expenditures?

A Yes.

Q What is that opinion?

A I agree that the proper use of healthcare will -- preventive healthcare does have potential for effect on the costs, overall costs of health and medical care.

Q Could you explain why that is?

A Well, to give you an example, if you can

avoid prematurity, you avoid a tremendous expense because premature infants remain in hospitals longer, even if they suffer no consequences, complications of the prematurity; whereas the full-term baby generally goes home within a day or two after birth.

Q Doctor, is it true that all children and all pregnant women do not utilize equally preventative healthcare?

A I agree with that.

Q Why is that?

A Sometimes it's the expense. Sometimes it's the understanding how healthcare will benefit you. Sometimes it's the access to healthcare. There are a number of variables. Sometimes it's educational level.

Q You're leading right into what I wanted to ask you, and that is, can you characterize the population that tends not to utilize preventive healthcare as much as it should?

A I think there are a number of populations that do that. There are the -- starting with the lower socioeconomic groups who are not covered by health insurance or government healthcare mandates; there are the yuppies who don't want to pay for health insurance, who feel that their bodies are

not subject to any risks to health at that particular stage in life; there are older people who feel that healthcare is unnecessary, that they've been healthy all their lives, and whatever the time to die occurs, they'll die.

So there are many different -- and there are religious groups who -- the Christian Scientists who don't believe that any form of healthcare is consistent with their religious beliefs. So I mean, there are many different groups for whom preventive care is not something that is a high priority for them.

Q Is the medicaid population or the public assistance population one of those groups?

A It is one of those, in general, yes.

Q Do you have an opinion as to why that is?

A It may be that they don't know how to use the healthcare system or they may not -- I'm unfamiliar with Mississippi. In California anybody who qualifies for assistance can receive very, very good healthcare, in fact, sometimes better healthcare than those who are in a more restrictive managed care environment.

Q Doctor, do you have an opinion as to

whether there is any difference in the utilization of preventive healthcare by the children of smokers compared to the children of nonsmokers?

A I don't.

Q You do not have an opinion?

A I don't have any knowledge of the issue.

Q We touched on this question earlier, but I want to go back to it briefly, and that is, do children from different socioeconomic groups have different risk factors for disease?

A Yes.

Q Does an increase in disease among a population necessarily result in an increase in the utilization of medical care?

A Could you restate that, please.

Q Yes.

Does an increase in disease among a population always lead to an increase in the utilization of medical care?

A I'd say, in general, that's true.

Q Doctor, what is the -- I take it that during your years of practice in pediatrics that you've also interacted with the parents of your patients; is that correct?

A Yes.

Q And would you agree that parents have a very important role in their children's health?

A Yes.

Q Are there consequences for a child's health of parental substance abuse?

A There can be, yes.

Q What might those consequences be?

A Well, in the very extreme inattention. And at the other extreme can be abuse. I mean. . .

Q But it might include inattention?

A Yes.

Q Do you have any understanding of the incidence of substance abuse among the medicaid population of Mississippi?

A No, I don't.

Q Do you have any understanding of the -- and I apologize if I've asked this question before. I don't think I've asked it quite like this.

Do you have any understanding of the medicaid expenditures in Mississippi for the children of smokers versus the children of nonsmokers?

A Can you sort of rephrase your question?

Q Sure.

A You're asking if I have any knowledge?

Q Yes.

A I don't.

Q For each of the -- I keep using the word "diseases" in reference to your 26(B)(4) Statement, and I'm not sure that you consider all of the health consequences that you described in that statement to be diseases.

A That's correct.

Q How would you -- what other term could I use to describe them?

A Well, it's -- your question is very broad.

Q I'm just trying to find a nomenclature to make it easier for us to talk --

A I don't think there's a single nomenclature. I think the exposure to cigarette smoke, if you're talking about the fetus, has a different consequence than perhaps the exposure to maternal smoke after delivery.

Q Can we use the term "health consequence," then?

A Okay. Again, I think it depends on the stage in fetal neonatal pediatric life, but there are a broad range of changes that occur in metabolism in association with exposure to cigarette smoke.

Q Well, if you'll permit me to do so, in

my own inarticulate way, I'll use the term "health consequence" to mean each of the things that you've described in your 26(B)(4) Statement.

My question is, for each of the health consequences that you've described in your 26(B)(4) Statement, would you agree that exposure to cigarette smoke is not a necessary predicate to that consequence?

A I don't agree with you.

Q You do not agree?

A (Witness replies by shaking head from side to side.)

Q Let me try to ask it a different way and see if your answer changes.

For each of the health consequences that you describe in your 26(B)(4) Statement, would you agree that cases occur among -- in individuals never exposed to cigarette smoke?

A I think I could agree if the semantics were different.

Q Would you try for me?

A Okay. I would agree that almost every observed change in the fetus, newborn infant, infant child may be caused by another stress, a different stimulus. I won't even use the term "stress."

Stimulus.

Q So you would agree, then, wouldn't you, that cigarette smoke is not a necessary cause for each of those changes?

A Yes. I haven't looked at each one and thought about it, but I would say, in general, I would agree with you.

Q Wouldn't you also agree that exposure to cigarette smoke in whatever form, either maternally or environmental tobacco smoke, is also not sufficient to bring about those diseases in every instance of exposure -- those health consequences in every instance?

A That's a very difficult question to answer.

Q Then I must have stated it wrong because I think it's an easy question if I can get it right.

A Okay.

Q The question I'm asking is wouldn't you agree that every mother who smokes does not deliver a low-birth-weight baby?

A I would agree to that as a general concept.

Q And wouldn't you agree that for all of the health consequences that you've described in your

statement they don't occur for every individual exposed to cigarette smoke?

A Given that there's a statistical range and within those statistical ranges thinking of standard deviation, having at least 5 percent of the population standing outside of that statistical variation, I would agree with you.

Q Well, wouldn't you agree that not every child exposed to environmental tobacco smoke develops otitis media?

A I would agree with that.

Q Wouldn't you agree that not every child exposed to environmental tobacco smoke develops a lower respiratory tract illness?

A That I would disagree with. Every child develops respiratory tract illness.

Q I mean attributable to environmental tobacco smoke.

A Oh, sure.

Q Wouldn't you agree that not every child exposed to environmental tobacco smoke develops an upper respiratory tract illness attributable to environmental tobacco smoke?

A I would agree with that.

Q Okay.

Wouldn't you agree that not every pregnant woman who smokes delivers prematurely?

A I agree.

Q Doctor, do you have any knowledge of where Mississippi ranks among the states with respect to per capita income?

A No.

Q If Mississippi ranked very low or last in per capita income among the states, what, in your opinion, would be the consequences for children's health in Mississippi?

A That's such a broad question, I couldn't answer.

Q You can't answer that question?

A No.

Q But you do agree that socioeconomic status has health consequences?

A Yes.

Q And the lower the socioeconomic status, the more likely it is for children to develop health consequences?

A Yes.

Q Doctor, what do infant mortality rates for a state tell you about the healthcare system in the state?

A Again, that's a very broad question.

Q Can you answer it?

A Yes. I think that the neonatal or perinatal mortality rates reflect a broad array of variables from access to healthcare to the socioeconomic status of the recipients of healthcare. The nutrition. I mean, there are a whole group of issues. Access.

Q Among those issues, do infant mortality rates have a meaning with respect to the quality -- or do they shed any light on the quality of medical care in the state?

A Again, it's a very broad question. Depending on access to healthcare, that is, assuming that everyone in the population had access to healthcare, it certainly would be -- if the mortality rate was very high, it would be a condemnation of the type of healthcare being obtained.

Q But, again, like some other situations we've discussed, it might be difficult to sort out what to attribute the mortality rates to?

A Yes. Exactly.

Q Doctor, do you have any knowledge of infant mortality rates in Mississippi?

A No.

Q What is the respiratory -- I may say this wrong -- syncytial, s-y-n-c-y-t-i-a-l, virus?

A RSV. I mentioned that term. It's a very common virus that can be misinterpreted, or it's one of the viruses that cause the common cold.

Q Did any of the studies that you reviewed in preparation for this deposition control for RSV infection?

A I don't believe they did, no.

Q Hasn't it been reported that RSV infection is strongly associated with a number of pulmonary consequences in children, such as bronchitis or pneumonia separate and apart from the environmental tobacco smoke?

A It's been associated with bronchialitis and pneumonia, yes.

Q Doctor, do you know whether -- let me ask this first for infants -- whether infants retain more or less of the particulate matter that they inhale in the range of .5 microns than adults do?

A It would depend upon the particulate matter.

Q How does that work?

A Well, for example, we've done some studies with aggregated albumen where it's taken up

in the lung; that is, that you can determine the permeability of the lung by looking at the uptake in the lung. In other words, the smaller the mean mass diameter of the particle, the farther into the airway the particle gets.

Q Let me ask the same question specific to cigarette particulate matter.

A Uh-huh. I don't really -- that's one of the areas I really haven't looked into.

Q You have not looked into that?

A No.

Q You haven't looked into it for children, either?

A No.

Q You may have answered this by telling me it depends upon the particulate matter, but it has been my understanding that, in fact, children retain less particulate matter in that range than adults do because of a combination of their respiratory rates and other factors.

Is that overly simplistic?

A Yes, it is, because, again, if you're talking about a six- or seven-year old, it's substantially different than a newborn infant or a premature, and I would -- I'd have to speculate on --

I know a lot about the development of the airway, but, as I said, I'd prefer not to speculate on issues that I don't have solid data on.

Q I think we all prefer that.

I know that you have told me that you've made no studies of the Mississippi population. There are a few more questions I'll ask --

A Uh-huh.

Q -- with respect to it, however, just to make sure the record is clear.

And one of them is, do you have any opinions as to the causes of pain and suffering among children in the Mississippi medicaid population?

A No.

Q Back to an epidemiologic question.

If parental smoking is correlated with a factor that is itself a risk factor for a certain disease in children, what are the consequences of failing to control for that factor in an epidemiologic study of children?

A Could you be more specific.

MR. PATRICK: I'm going to object to the question on form.

MR. FURR: Okay.

Q If parental smoking is correlated with

socioeconomic status, what is the consequence of failing to control for socioeconomic status in a study of children and a disease that socioeconomic status is itself correlated with?

A In what way are you talking about controlling for the smoking?

Q I'm sorry. Let me try it a different way.

A I may be thinking of a different way of controlling than you are but. . .

Q What I'm trying to ask you is a question about confounding, and wouldn't you agree that factors that are correlated statistically with parental smoking that are also correlated statistically with the incidence of a disease that you're attempting to examine must be controlled with confounders in a study of that disease in children?

A Well, there are two ways of controlling; one is you could ask or measure the number of cigarettes someone's smoking; and another, you could look for a specific impact of the smoking in the individual being studied.

Q I'll try it again later.

A No. Maybe I -- what I'm saying is that, if you ask somebody if they engage in a particular

habit, you get a variable response, either none or somewhere on a sliding scale, and that is subjective.

However, if -- let's take phenobarbital again, and they say they take phenobarbital. But if you measure in the plasma, their plasma, and you find no phenobarbital, it's irrelevant whether they tell you they do or they don't because we know phenobarbital has a long half-life, and if they've taken it within the last three days, you're going to find at least half the amount that they took still in the plasma.

So there would be different ways for controlling for phenobarbital use. One would be asking, but a much better way would be measuring the amount of phenobarbital in the person's serum.

Q Let me try it this way.

If I want to study the incidence of otitis media in the children of smokers, and I know that parental smoking is also correlated with socioeconomic status, what are the consequences of failing to control for socioeconomic status in my study of otitis media?

A I would think that it would have an impact on the study.

Q Would you agree that it would confound

the results of the study?

A I agree.

MR. FURR: Let's take a five-minute break.

(Recess from 11:10 a.m. to 11:19 a.m.)

BY MR. FURR:

Q Doctor, did you agree earlier that the demographics and life-style of children are largely determined by their parents?

A Yes.

Q Are you familiar with the differences in demographics and life-styles of smokers versus nonsmokers?

A No.

Q Not at all?

A You'd have to be more specific with regard to the demographics and life-style. Life-style is sort of a broad term.

Q If someone is a smoker, are they more likely to be of average or below average socioeconomic status?

A I think statistically they'd be of lower socioeconomic status.

Q Are you familiar with the dietary differences between smokers and nonsmokers?

A No.

Q Are you familiar with the difference in educational level between smokers and nonsmokers?

A Statistically, I am aware that smokers are generally considered to be of lower educational status.

Q Do you have an opinion as to whether you are likely to be a member of a minority if you are a smoker?

A No. I couldn't give you the data on that.

Q Have you ever performed any studies of the difference in the life-styles of the children of smokers versus the children of nonsmokers?

A No.

MR. PATRICK: Let me object. Life-styles of the children or life-styles of the smokers?

MR. FURR: Of the children.

THE WITNESS: No.

BY MR. FURR:

Q Have you read any studies examining that question?

A If you could be more specific what you mean by "life-style," maybe I could help you.

Q Are you familiar with any studies of the socioeconomic status of the children of smokers

versus that of nonsmokers?

A I already answered that. Yes, I am.

Q I'm sorry. I don't recall asking.

What was your answer?

A I said that, in general, the statistical data suggests that the children of smokers are children of people of lower socioeconomic status.

Q And did I also ask you this question for nutrition?

A I don't remember.

Q Do you have any opinions as to nutrition of children of smokers versus that of nonsmokers?

A Do I have any opinions?

Q Yes.

A No.

Q I want to turn to a specific health consequence, and that is premature delivery.

A Uh-huh.

Q And I want to ask you what your opinions are with respect to maternal smoking and premature delivery.

A Could you be more specific on that? What aspect?

Q Is there a link between maternal smoking and premature delivery?

A Yes. I think there has been a linkage on several levels.

Q Could you explain those?

A Well, first of all, the studies that I reviewed suggest that the lower the exposure of the mother to cigarette smoking, the less the risk is to the fetus of delivering early.

I could go on if you'd like.

Q Please. Please.

A Second of all, that at higher levels of exposure, the risk is at two levels; one of an obstetric nature with regard to increased risk of bleeding or abnormal placentation, that is, either abruptia placenta, where the placenta separates early and there's bleeding either from the maternal or fetal side, or placenta previa, where the attachment of the placenta to the uterus is at a disadvantaged site, and bleeding is often a consequence and vaginal delivery is complicated, both of which may lead to premature delivery.

The other is that there is an association between premature rupture of the membranes, as well, which may cause premature delivery but not in every instance.

Q Can you describe the dose response for

these consequences? What level of maternal smoking are we talking about, for example?

A There are so many different studies that --

Q Your opinion is what I'm seeking.

A I would think that, from the literature, it would be at a higher level of smoking, greater than ten cigarettes a day. There are things -- I'd have to look back at the articles. I'm saying this from my memory of the data that I reviewed.

Q From maternal smoking of less than ten cigarettes per day, what are the consequences with respect to premature delivery?

A I don't think I saw any data to convince me that there is a positive association.

Q For more than ten cigarettes per day, what is the size of the association?

A As I said, I thought some of the studies reported significant association, but that I can't give you the specific values. I'd have to review that.

Q Do you have an opinion as to what -- the range that the value would fall in?

A Again, this is surmise. I believe that the odds ratios were two and a half or somewhere

around there.

Q And what studies do you base this opinion on?

A I'd have to go back and look. I'd have to look. It would be -- I don't have that here.

Q Well, we earlier discussed that risk observed in epidemiologic studies apply only to populations that are being studied into analogous populations.

Does the risk that you've just -- do you know what population the risk that you have just provided to us of 2.5 applies to?

A No. I'd have to, again, go through my notes.

Q Do you have an opinion as to what the risk of smoking greater than ten cigarettes -- relative risk of smoking more than ten cigarettes per day would be for premature delivery in the Mississippi medicaid population?

A No.

Q Doctor, do you have an opinion as to what the most important step is that could be taken to decrease the incidence of premature delivery in the Mississippi medicaid population?

A As I said, I'm not a student of medicine

in Mississippi, so I really couldn't comment.

Q Do you have an opinion as to the effect on the incidence of premature delivery in Mississippi that would be achieved by the elimination of maternal smoking?

A I have no idea.

Q In your review of the literature, did you come across any studies that found no link between cigarette smoking and premature delivery?

A Yes.

Q Do you recall which studies those were?

A No.

Q I take it from your opinion that you have rejected those studies, and my question is, what is your basis for rejecting those studies?

A I think your assumption is incorrect.

Q Could you explain?

A Yes. I think of all the issues that I've looked at, prematurity is one of the more difficult ones to study. And that the studies need to be perspective, and they need to take into account the combination of the length of time from conception, as well as the incidence of birthweights less than 2500 grams.

To clarify, if a baby is less than

2500 grams, which is the usual size of the infant at full-term birth or the expected minimal -- if the baby is born less than that, it doesn't mean that the baby is premature. And some of the studies were as -- well, they may have been done well, but they didn't convince me that that had been taken into account.

Q Doctor, I thought that the -- whether a baby is delivered premature or not was based primarily upon the weeks of gestation?

A That's what I'm saying, that some of the studies actually reported weight rather than gestation and corroboration of gestation.

Q Returning to something we talked about earlier, and my question is, how, in your mind, did you harmonize those studies that did not report an effect from maternal smoking on the incidence of premature delivery and those that did?

A I think I tried to explain. I think that there is a high amount of controversy that still persists over whether prematurity, per se, is impacted substantially by cigarette smoking. And the reasons for that are probably multiple, some of which we've talked about. But others depend on the linkage between the length of time from conception and the

assessment of the babies' gestational age by physically examining the birth.

Q Do you have an opinion with reasonable medical certainty as to whether maternal smoking has an impact on premature delivery, per se?

A Yes.

Q What is that opinion?

A I would think that the risk is probably greater, but I couldn't give you concrete data on how much of the risk is due specifically to cigarette smoke. It's a factor, but there are many factors.

Q Let's talk about some of those factors. Mark this as E, please.

(Defendant's Exhibit E was marked for identification and is attached hereto.)

BY MR. FURR:

Q Doctor, are you familiar with an instrument known as the Creasy scale that is used for assessing prematurity -- excuse me -- for assessing the risk of premature delivery?

A Not until now. I know Bob Creasy very well, but. . .

Q Are you aware, Doctor, that a number of instruments have been developed for use in assessing

the risk of prematurity?

A Yes.

Q You've been handed Platzker Exhibit E for identification, which contains a table on Page 26 labeled the "Creasy Scale."

A Uh-huh.

Q I take it that you're not familiar with this scale?

A No.

Q Let's work through the table and let me ask you a few questions.

Would you agree, Doctor, that having two children in the home is a risk factor for premature delivery?

A I can't really comment on that.

Q You don't know?

A I don't know.

Q Would you agree that low socioeconomic status is a risk factor for premature delivery?

A Yes.

Q Would you agree that maternal age of under 20 years or older than 40 years is a risk factor for premature delivery?

A I don't totally agree with that.

Q You do not agree with that?

A No.

Q Why is that?

A The data that is present suggests that those younger than 20, if in good follow-up following conception, that is, that they receive regular healthcare, in most of those under 20 are at no greater risk for premature delivery than those over 20, the age used as a single factor.

Q Do you agree that patients -- that mother's over 40 years, maternal age of over 40 years is a risk factor?

A Yes.

Q Do you agree that having had an abortion in less than a year since the last birth is a risk factor for premature delivery?

A I'd have to say that I've heard that multiple times, but I'm not aware of how well-proven it is.

Q Do you agree that working outside the home is a risk factor for premature delivery?

A No.

Q Do you agree that heavy work is a risk factor for premature delivery?

A Again, I would agree with that on the basis that it sounds reasonable, but I'm unaware of

the studies to document that.

Q Do you agree that experiencing unusual fatigue during the pregnancy is a risk factor for premature delivery?

A I'd answer that the same way I answered the last question; sounds reasonable, but I'm not certain of the recent studies that confirm that.

Q Do you agree in a maternal weight gain of less than 13 kilograms by 32 weeks' gestation is a risk factor for prematurity?

A Again, I'd have to answer that the same way.

Q Do you agree that a finding of albumen in the urine is a risk factor for prematurity?

A Yes.

Q Do you agree that hypertension is a risk factor for premature delivery?

A Yes.

Q Do you agree that a finding of bacteria in the urine is a risk factor for premature delivery?

A Yes.

Q Do you agree that breech at 32 weeks is a risk factor for premature delivery?

A Again, that's one of the things that I believe, but I'm not aware of the studies to document

that.

Q Do you believe that maternal weight loss of two kilograms during the course of the pregnancy is a risk factor?

A The same issue.

Q Do you believe that a febrile illness during the course of pregnancy is a risk factor?

A Again, I would think that sounds reasonable, but I'm not aware of the studies to document that.

Q What does m-e-t-r-o-r-r-h-a-g-i-a mean?

A That means bleeding.

Q How do you say that word?

A Metrorrhagia.

Q Do you believe that metrorrhagia after 12 weeks' gestation is a risk factor of premature delivery?

A Yes.

Q Do you believe that effacement is a risk factor?

A Yes.

Q Dilatation?

A Yes.

Q Do you believe that uterine irritability is a risk factor?

A Yes.

Q Do you believe that placenta previa --

A Previa.

Q -- previa is a risk factor?

A Yes.

Q Do you believe that hydramnios is a risk factor for premature delivery?

A Yes.

Q Do you believe that being pregnant with twins is a risk factor for premature delivery?

A Yes.

Q Do you believe that having abdominal surgery during the course of pregnancy is a risk factor?

A That's one area I'm unaware of the literature, but it sounds quite reasonable.

Q Doctor, I'll ask you to take a look at that scale and note the left-hand column of the Creasy Scale on Page 26. And there you see an assignment of points to various factors.

A Uh-huh.

Q Do you understand the way those points are used in this instrument?

A I haven't read the papers, so I'm certainly not aware of how it's been used. I suppose

that if I look down here, it says that medium risk is a higher point score than a low risk.

Q Based upon that assumption, do you have any disagreement with the weight that has been assigned risk factors in this table with respect to the importance for predicting premature delivery?

A I'm really not qualified to make that assessment.

Q Okay. That's fine.

Doctor, do you have an opinion as to what the most important factors in the Mississippi medicaid population are for predicting premature delivery?

A No.

Q Doctor, isn't it true that even using these scales that have been developed, including the Creasy Scale, that for a first pregnancy only about 60 percent of the premature deliveries can be predicted?

A That sounds like a good number, but --

Q Do you have any familiarity with that issue?

A I can't really comment on the percentage.

Q Would you agree that something less than

a hundred percent of premature deliveries can be predicted using these scales.

A What I agreed to was that I would agree that with a first pregnancy it's very difficult to predict the risk of prematurity, but I'm unable to put a particular percent on how many of the instances we can predict -- in which we can predict prematurity.

Q Why is it difficult to predict the risk of premature delivery with a first pregnancy using these scales?

A Because we're totally unaware of some of the maternal obstetrical factors that can occur in a particular individual.

Q And are we also unaware of the factors that bring about those maternal obstetrical characteristics?

A I think some we understand and others we don't. That is, if a mother has hypertension prior to pregnancy, she is at greater risk for hypertension with the pregnancy, and she may have to be delivered earlier. That's just one factor.

Q Would you also agree that there must be other risk factors for premature delivery that we're simply unaware of?

A Again, I haven't, like Bob Creasy, studied the issue of prematurity to the extent that I can exclude other factors that might have occurred that might be out there but haven't been identified.

Q Are you aware, Doctor, of different rates of premature delivery in different populations?

A Yes.

Q And are you aware as to the correlation in different -- strike that.

Are you aware of different smoking rates in those same populations?

A Perhaps you could clarify.

Q Let me reask the question.

Are you aware that there are populations with much lower rates of premature delivery and much higher smoking rates than in the U.S. population?

A That there are populations with much higher smoking rates and lower --

Q The other way around.

Are you aware that there are populations that have much lower rates of premature delivery and much higher smoking rates?

A That's what I said.

Q I'm sorry.

A Yes.

Q And how would you explain that?

A I haven't studied that, but again, it would depend on the dose response, that is, how much of whatever incites premature delivery in, for example, cigarette smoke. Arbitrarily, we'll talk about cigarette smoke. It's in the person, that is, that in a particular population, if they were able to metabolize nicotine, et cetera, more rapidly, that they may have sustained lower rates, lower blood levels, tissue levels of whatever harmful substances are in cigarette smoke than other populations. So they could smoke more and sustain a lower incidence of uterine irritability, effacement, dilatation, whatever, as risk factors for hypertension.

Q Doctor, are we back to the point that it really is just very difficult to draw inferences across populations?

A No. I think if you have a very homogeneous population, you can make some predictions; that is, that if you want to use, for instance, Chinese in California have an incredibly low rate of prematurity.

Now, I don't know unless we studied it, whether the same would be true of China, Chinese living in China. If so, then the studies in one

population might be analogous to the other. It's how well-controlled your population is.

Q Doctor, if an individual case of premature delivery is presented to you, is there anything that you can rely upon to link an individual case of premature delivery to cigarette smoke?

A No.

Q You simply can't tell?

A No. I mean, the scenario that you've given me, a patient comes in and it's a premature baby, is this due to smoke? There's no way of telling with the information available.

Q There's no test that could be performed?

A Well, I mean, you could perform tests. But other than to say that the risks are possible that it's due to this or that, it's often unable, impossible in a particular case to attribute to a particular stimulus the cause of prematurity.

Q I now would like to talk about low birthweight and any opinions that you might have with respect to maternal smoking and the incidence of low birthweight.

Could you give us your opinions, please.

A I think there is fairly uniform agreement that there are some substantial reductions

in birthweight depending on the amount of smoking by the mother.

Q Can you describe for us your understanding of that dose response-curve?

A Yeah. Most of the studies I've read, I think -- and they still continue to publish studies on this -- suggest that less than ten cigarettes a day, the amount of reduction in birthweight is usually less than 200 grams. It's the number that seems to come up all the time is about 180.

Q And what's the risk of having an infant of less -- excuse me -- a decrement in birthweight of 180 grams for a woman who smokes less than ten cigarettes per day?

Is there a relative risk that you can attach to this?

A Well, I couldn't give you the specific risk. I'd have to look at analysis of all the studies and tell you what the risks are, but statistically that would be the amount of reduction in body weight for that amount of smoking.

Q What about for more than ten cigarettes per day?

A 10 to 19 it's usually about 340, 350 grams, in that range.

Q More than 19?

A I can't really tell you how much. I don't -- I really haven't seen many studies that reflect on that amount of smoke.

Q Doctor, I take it that you have no opinion as to what the dose-response relationship might look like in a Mississippi medicaid population?

A No.

Q What is the significance of a 200 decrement in birthweight?

A Could you be more specific.

Q What are the medical or health consequences for the infant of a 200-gram decrement in birthweight?

A Well, I think the decrement in weight probably means very little in of itself in most cases.

Q Does a 200-gram decrement in birthweight lead to any additional medical expenditures in most cases?

A The weight reduction alone, probably, I can't comment on whether it leads to any increased expenses. I certainly didn't see a great deal in the literature commenting on that.

Q What is the health consequence of a 340

or 350 decrement in birthweight, gram decrement in birthweight?

A I think if we understand that the weight is just one concrete measurable objective evidence of a loss in potential and that may be shared by all the organs of the body, then there may be significant long-term consequences.

Q "There may be"?

A (Witness replies by nodding head up and down.)

Q Are there any specific medical expenditures that you can attach to a 350-gram decrement at birthweight?

A Medical expenditures?

Q Yes.

A With regard to the weight alone, it would depend upon what the baby's weight is with the loss in the 340, 350 grams. It may be no greater expense, or it could be substantial depending on what the impact was on the baby's medical condition.

Q With respect to the weight alone, do you have an opinion as to what weight the 350-gram decrement becomes important with respect to medical expenditures, what infant weight?

A I mean, even if the baby has no other

definable problems, if the baby's weight was less than two kilograms at birth, generally, it would mean extra days in the hospital.

Q But above two kilograms at birth, a 350-gram decrement --

A With no other problems, it may not keep the baby in the hospital any longer.

Q I want to talk with you now about other risk factors for low birthweight.

Would you agree that premature delivery is a risk factor for low birthweight?

A Yes.

Q So would you, then, agree that all of the factors that we discussed earlier and that you agreed are risk factors for premature delivery are also risk factors for low birthweight?

A You mean the factors in the risk chart of Creasy?

Q Yes.

A I would have to go over each one and really comment on them.

Q Do you believe that socioeconomic status is a risk factor for low birthweight?

A I suppose so.

Q Are you aware it's been reported to be a

risk factor for low birthweight?

A Yes.

Q You don't have any contradictory evidence, do you?

A No.

Q Do you believe that the order of birth of the infant is a risk factor for low birthweight?

A Order of birth?

Q Yes.

A Could you be more specific.

Q Isn't it true that being the first-born to a mother is a risk factor for low birthweight?

A No.

Q Is it true that being the last-born to a mother is a risk factor for low birthweight?

A No.

Q Is there a relationship between order of birth and low birthweight?

A I believe, from -- my understanding is that the first baby of any mother is lower in birthweight than babies born subsequently if there is a separation of pregnancies, at least two-years' separation.

Q Is marital status of the mother a risk factor for low birthweight?

A Per se?

Q Per se.

A No.

Q Are you aware that it has been reported to be a risk factor for low birthweight?

A Yes, I have.

Q And why do you reject that?

A Because I think marital status divorced from other variables, such as socioeconomic and racial issues, is invalid.

Q That was an interesting play on words, by the way; marital status of the mother "divorced from. . ."

Do you believe that maternal age under 17 is a risk factor for low birthweight?

A Yes and no. Yes in that it's less likely that a 17-year-old, someone less than 17 would be in healthcare followed as closely, perhaps.

No because, if they are in fairly good medical surveillance, the risks have been shown not to be any greater than any other population, age group.

Q You agree, then, that the failure to use prenatal care is a risk factor for low birthweight?

A Yes.

Q Do you agree that maternal age over 34 is a risk factor for low birthweight?

A Yes.

Q Do you agree that the educational achievements of the mother are correlated with birthweight?

A I'd prefer that you reword the question.

Q Is there a relationship between mother's educational status and birthweight?

A Yes.

Q What is that relationship?

A That is that mothers who don't graduate high school tend to have lower-birth-weight babies.

Q Why is that?

A I think that's not linked specifically to -- and this is my own opinion -- to educational achievement as much as it's related to socioeconomic status.

Q Do you agree that the nutritional status of the mother is a risk factor for low birthweight?

A Yes, poor nutritional status.

Q Poor nutritional status.

Do you agree that infectious processes during pregnancy are a risk factor for low birthweight?

A That's a hard question to answer in the way you've asked it.

Q Is there a correlation between infection during pregnancy, infections in the mother during pregnancy and birthweight?

A Specific infections, yes.

Q What infections are those?

A Urinary tract infections, which is a major risk factor for prematurity. Other infections which may affect the fetus directly, that is, infect the fetus, also tend to cause smaller than appropriate for gestation-age babies.

Q What infections might those be?

A There's a broad group of infections called the Torch Group, T-o-r-c-h, which include sexually transmitted disease, a disorder called Toxic plasmosis, Cytomegaloviral disease, Syphilis, being a sexually transmitted disease, Herpes simplex, which can lead to infants who are also infected, some born prematurely, but even those born at term are lower birthweight tend to be lower birthweight.

Q You anticipated my next question.

Are those Torch factors that you've just listed for us also risk factors for premature

delivery?

A Many are.

Q Many are?

A Several are, yes.

Q Which are not?

A I'm not sure that toxic plasmosis is. Congenital rubella I don't believe has a high incidence of prematurity associated with it.

Q Is mothers' use of alcohol during pregnancy a risk factor for low birthweight?

A Yes.

Q Is mothers' use of illegal drugs a risk factor for low birthweight?

A Yes.

Q What drugs would that include?

A Well, speed, amphetamines would include heroin, cocaine.

Q Would it include crack cocaine?

A Well, it's just another variation on the same thing, but it's cocaine.

Q Is poor weight gain during pregnancy a risk factor for low birthweight?

A Yes.

Q Are there maternal genetic factors that are risk factors for low birthweight?

A Yes.

Q What are those?

A Genetic factors?

Q Yes, sir.

A Well, mother's small stature is the primary one that would be a major risk factor for low birthweight, but there are others.

Q Is mother's weight also a risk factor for low birthweight?

A I think it's probably weight gain rather than her weight, per se, weight gain during pregnancy.

Q Is unusual fatigue during the pregnancy a risk factor for low birthweight?

A I can't comment on that. I'm not aware of the literature.

Q Is a finding of albumin in the urine a risk factor for low birthweight?

A Yes.

Q Is hypertension in the mother a risk factor for low birthweight?

A Yes.

Q Is a weight loss of two kilograms during pregnancy a risk factor for low birthweight?

A Again, I would think it would be, but I

couldn't quote the literature.

Q Is effacement a risk factor for low birthweight?

A For prematurity. It has nothing to do with --

Q Low birthweight?

A -- low birthweight, not primarily, but secondarily because of prematurity.

Q As we talked about, prematurity itself is a risk factor?

A Right.

Q I assume that having twins is a risk factor for low birthweight?

A Yes.

Q Or other multiple pregnancies?

A Well, having twins is associated usually with premature delivery, and therefore, low birthweight.

Q Are there other risk factors for premature delivery that I have not asked you about?

MR. PATRICK: Premature delivery or both birthweight --

MR. FURR: Premature delivery.

Q I'm sorry. I forgot to ask you when we were talking about premature delivery.

A I think you covered most of them; premature rupture of the membranes and bleeding we didn't discuss, specifically, but I think both of those would be included.

Q What do you believe to be the most important risk factors for premature delivery?

A I couldn't really say any one particular risk factor.

Q Are there other risk factors for low birthweight that we have not talked about?

A Gee, I can't think of any.

Q Which of the factors that we've talked about do you consider to be the most important risk factors for low birthweight?

A One factor?

Q As many factors as you think are in the level that are most important.

A Well, I would think starting with the mother, a woman who tends to have infants with low birthweight, that would be a risk factor; women who tend to dilate and efface early, again, because of prematurity, that would be a risk factor; women who have very poor nutrition, protein nutrition, especially, would be a risk factor; women who smoke, it's a risk factor; women with hypertension is a risk

factor. I mean, there are many risk factors.

Q Doctor, are you aware that it has been reported that among women who smoke -- I should say -- strike that.

Are you aware that it has been reported that for the infants of women who smoke, that any decrement in birthweight is recovered by six months of life?

A The data I've said I've reviewed suggests that it's variable sometime between six months and a year, but there's catch-up growth.

Q Between six months and a year, you believe that there is catch-up growth?

A Catch-up weight gain. I haven't seen data on the other measurements of growth.

MR. PATRICK: I don't know what you were planning to do about taking a break for lunch or how much longer you may have, or do you intend to go until 5:00, which is fine?

MR. FURR: I think we probably intend to go until 5:00, but whatever is most convenient for lunch. And now is fine with me, if that's what you'd like to do.

MR. PATRICK: Whatever the doctor wants to do.

Why don't we go ahead and break now.

MR. FURR: Okay.

MR. PATRICK: We'll try to be back here in an hour or sooner than that?

MR. FURR: Let's be here in an hour.

(At 12:10 p.m. a luncheon recess was taken, the proceedings to be resumed at 1:10 p.m.)

AFTERNOON SESSION

(At 1:20 p.m. the proceedings were resumed at the same place.)

EXAMINATION (Resumed)

BY MR. FURR:

Q Doctor, let's try to tie up the low birthweight topic. I'm going to ask you some questions about low birthweight that I hope were similar to those that I already asked you about earlier.

The first is, is there anything about an individual case in which an infant has been born at low birthweight that would allow you to link that individual case to cigarette smoking by the mother?

A No.

Q Next, I tried to ask you this question earlier, and I believe I stumbled. But is it possible to attach a relative risk or odds ratio to the likelihood of having a low-birthweight infant for a mother who smokes ten or less cigarettes per day?

A Various studies have shown odds ratios, but they've been in various different populations so that it varies.

Q Do you have an opinion as to what that odds ratio would be in the Mississippi medicaid population?

A No.

Q The same question for more than ten cigarettes per day; do you have an opinion as to what the odds ratio would be for a low-birthweight delivery in a mother who smokes more than ten cigarettes per day in the Mississippi medicaid population?

A No, I don't.

Q What are the most important steps that could be taken to decrease the incidence of low-birthweight infants in the Mississippi medicaid population?

A That would depend on the population, and I'm not sufficiently familiar with the various

variables in terms of pregnant women in Mississippi.

Q Do you have an opinion as to what the impact on the incidence of low-birthweight infants in the Mississippi medicaid population would be if smoking was banned tomorrow?

A I think there would be an impact, but I'm not able to say what the impact would be.

Q You're not able to quantify it?

A That's right.

Q Are you able to quantify what the impact on medical expenditures under the medicaid program in Mississippi would be?

A No.

Q I'd like to talk to you now about otitis media in environmental tobacco smoke.

Do you have an opinion with respect to the consequence of environmental tobacco smoke exposure for the incidence of otitis media?

A I think it increases the risk in infants who sustain upper respiratory tract infections.

Q I'm sorry. I didn't hear you.

A I think it increases the risks in those infants experiencing an upper respiratory tract infection.

Q I'm confused here. It's my lack of

knowledge.

Is otitis media the upper respiratory tract infection you're describing, or is it another upper respiratory tract infection?

A Otitis media is a frequent complication of upper respiratory tract infection, albeit a common cold or other respiratory viral infection in children.

Q And what studies do you base your opinion on?

A Well, there are numerous studies that have been published suggesting that children in settings in which the parents smoke are at greater risk for otitis than situations in which parents don't smoke.

Q Are these studies that you uncovered in your literature review?

A Yes.

Q In the course of that review, did you also uncover studies finding no association between parental smoking status and the incidence of otitis media?

A No, I didn't.

Q You did not find any?

A (Witness replies by shaking head from

side to side.)

Q Can you describe a dose response for ETS exposure of parental smoking and the incidence of otitis media?

A No, I can't, mainly because I don't remember whether there was in any of these studies substantial dose-response data. I'd have to check my notes.

Q Can you attach an odds ratio or relative risk for the development of otitis media from parental smoking?

A Specific odds ratio?

Q Yes.

A No.

Q Is the association between parental smoking and otitis media incidence limited to maternal smoking, or does it also extend to paternal smoking?

A It's most likely -- it's most closely associated with maternal smoking, that is that if the father smokes and the mother doesn't smoke, there isn't an increased risk. But if both -- if the mother smokes and the father smokes, there seems to be a greater risk of otitis in those infants who have a cold or other respiratory viral illness.

Q Did I understand you to say that there is no increased risk if only father smokes?

A Yes.

Q Why is that?

A In most homes the mother is the primary caretaker. In most homes, again, that have been studied, the father goes to work; the mother stays at home.

In addition, in the routine caregiving, mothers have to pick up their babies, and the babies are close enough to the clothes where any impregnation of cigarette smoke in the clothes ends up being inhaled by the baby.

Q So is this primarily a dose phenomenon, then?

A It can be a dose phenomenon. There isn't any data on the size of the space, the amount of contribution of inhaling, absorbing it from the clothes versus inhaling it from the air, the environment.

Q Doctor, I take it, then, that you can't attach a relative risk for otitis media to maternal smoking in the Mississippi medicaid population?

A No.

Q What are other risk factors for otitis

media?

A Well, upper respiratory tract infection is particularly at risk. Because of the anatomy of the upper airway of the infant, there are also -- once a child has had a single episode of otitis media, there is a heightened risk for further episodes due to the injury that may be caused.

There are other diseases for which otitis media is a natural complication.

Q What diseases are those?

A Well, any child with immune dysfunction, such as children who have cancer or are on cancer chemotherapy where their own host defenses are aberrant; children who are born with congenital hypogamoglobin anemia decrease in the humoral immunity of the child; infants born with a condition called De George syndrome in which the lymphocyte population, those cells that are called in to augment humoral immunity, are defective.

There are many different other causes of host defense alterations. Infants who are born with respiratory disease and develop a condition called bronchopulmonary dysplasia or chronic lung disease of infancy are at particular risk for otitis, as well.

Q It's a special research interest of

yours, isn't it?

A It's one of them, yeah, uh-huh.

Q Can you think of other risk factors for otitis media as you sit here?

A Well, we were talking about anything that causes inflammatory changes in the upper airway can lead to increased risk for otitis.

Q What might that be?

A Inhaling any noxious substances; cigarette smoke, ammonia fumes.

Q General air pollution?

A I haven't seen any data on air pollution.

Q Is race a risk factor for the incidence of otitis media?

A Not specifically unless there are other factors involved.

Q Is parental socioeconomic status a risk factor for the development of otitis media?

A Not alone, no.

Q Not alone?

A (Witness replies by shaking head from side to side.)

Q Are you aware that it's been reported to be a risk factor?

A Well, I'm saying that race, specifically, without taking into account other risk factors that might be associated with race hasn't been -- just race alone hasn't been taken into account.

Q So race may be a surrogate for other risk factors?

A Exactly.

Q Just the socioeconomic may be a surrogate for other risk factors?

A Right.

Q We don't know what all those factors may be; is that correct?

A That's probably true.

Q Does otitis media vary seasonally?

A Yes.

Q Why is that?

A Well, in most temperate climates, the frequency of significant respiratory illness seems to be concentrated in the winter months, fall and winter months, so that since there's an association between otitis media as a complication of upper respiratory tract infection, they go hand in hand.

Q Is the failure to breast feed an infant a risk factor for otitis media?

A Not primarily.

Q When you say "not primarily," I'm not sure I understand.

A The association there is that infants who are breast-fed have a lower risk for upper respiratory tract infection in the first year after birth so that since -- at least in my mind, I've made the association between otitis media being a complication of upper respiratory tract illness. If the mother breast-feeds and there's a lower risk for upper respiratory tract illness, then there'd be a lower risk secondarily for otitis media.

Q When you say "not primarily," you mean -- do you mean not directly?

A Not directly, yes.

Q But you would agree that the failure to breast-feed is linked statistically with the incidence of otitis media in the first year, wouldn't you?

A Yes.

Q Are you familiar with the characteristics of populations and their tendencies to breast-feed?

A You'd have to be more specific.

Q Does the incidence of breast-feeding

vary across different populations in this country?

A Yes.

Q Could you describe that for us, please.

A It's changed with time; that is that before the early '70s, breast-feeding had fallen into disrepute. Ever since the evolution of artificial formulas in the late '40s, early '50s, there have been an increasing number of, especially educated women, who had forfeited their opportunity to breast-feed in favor of artificial formulas.

In the '70s or late -- mid/late '60s, with the increasing interest in a healthy life-style, breast-feeding came back into vogue, and while initially it -- again, I'd have to speak of California since I've spent most of my professional life in California, but by the mid-'70s or late '60s, the upper socioeconomic groups, educated groups were all adopting breast-feeding.

By the mid-to-late '70s, this was a general trend. A hospital next to one in which I work, which has a fairly low socioeconomic group delivering there, had over 65 percent of the mothers selecting breast-feeding. This is beginning to change, at least in California, where with more mothers working and probably for other reasons fewer

mothers are breast-feeding.

Q Has low humidity been statistically linked with the incidence of otitis media?

A Gee, I'm not really aware of that as a major issue.

Q Is the presence of siblings with infectious diseases a risk factor for otitis media?

A Siblings with upper respiratory tract infections, yes.

Q Is RSV a risk factor for otitis media?

A Yes.

Q Are there other risk factors that you can think of that we haven't discussed?

A Yeah. There are other illnesses that have as a frequent complication otitis media. Rubeola, the seven-day measles, has as a very frequent complication to it otitis media.

Q Are there differences in the risk factors for recurrent otitis media and nonrecurrent otitis media.

A Are there risk factors?

Q Are there differences in the risk factors for those two diseases?

A Well, I think there are. I mean, for recurrent otitis media, the major risk factor is the

presence of an initial infection that sets the stage for an inflammatory change. If the child sustains another respiratory tract infection before the inflammatory response from the first infection resolves, then they're at risk for another one. But there are many risk factors for recurrence.

Q Am I correct in assuming that you've treated a lot of otitis media in your career?

A Yes, I have.

Q When a case of otitis media comes into your office, is there anything about that patient that would allow to you link it to exposure to tobacco smoke?

A No.

Q Earlier you said that you came across no studies that failed to find a relationship between parental smoking status and the incidence of otitis media; is that correct?

A Yes.

Q If you learned, in fact, that about two-thirds of the studies published in the peer review literature had failed to make such a finding, would that alter any of your opinions that you have expressed?

A Oh, I'd be happy to look at those

studies and comment on them.

Q But you'd have to examine the studies?

A Right.

Q Again, this is something we've talked about before. But if, in fact, that were the case and that there were studies that failed to find a link between parental smoking status and the incidence of otitis media, what process would you go about in trying to harmonize those results to reach your conclusion?

A Well, I think that's a reasonable task. One would be to look at the nature of the study to see how well controlled it was, that is, looking at an index group of infants of smokers versus an index group of infants of nonsmokers, looking at the other confounding variables such as whether the babies have been born prematurely, whether they had had respiratory distress syndrome, pulmonary dysplasia; look at both groups and see whether they were comparable.

Q So you would attempt to assess the quality of the studies?

A Right. Exactly.

Q And one of the chief factors in your assessment of the quality of the studies would be the

degree to which other variables have been controlled in the studies?

A Exactly.

Q Do you have an opinion what the impact on the incidence of otitis media in the Mississippi medicaid population would be if smoking were banned tomorrow?

A No.

Q I want to ask you some questions about not the induction of asthma but the exacerbation of asthma or the bringing on of an asthmatic episode.

Do you have any opinions with respect to environmental tobacco smoke exposure and the occurrence of asthmatic episode in asthmatic children?

A Yes.

Q What are those opinions?

A I think that being exposed to environmental tobacco smoke increases the risk of causing an exacerbation of asthma.

Q Are there any particular studies that you rely on for that opinion?

A Well, there are many studies from very good laboratories sort of confirming that association.

Q Can you describe those response relationships for environmental tobacco smoke exposure in the exacerbation of an asthmatic particular attack?

A No.

Q Can you ascribe an odds ratio to ETS exposure in the induction of an asthmatic attack?

A Not offhand, no.

Q I take it you would not be able to do so for the Mississippi medicaid population?

A No.

Q Do you treat a lot of cases of asthma?

A I've treated a number of cases of asthma.

Q If a child presents or is having a recurring asthma attack, is that called "status asthmaticus"?

A No. Status asthmaticus is a very distinct symptom of intractable asthma, that is, asthma that fails to respond to conventional intervention.

Q If a child presents to your office in the throes of an asthmatic attack, it there anything about that child that would allow you to link the attack to exposure to cigarette smoke?

A Only historically.

Q Only by history?

A Right.

Q No physical finding?

A No.

Q No laboratory test finding?

A No.

Q So you would attempt to draw an inference based upon the history that you received?

A Uh-huh.

Q What are other risk factors for induction of an asthmatic attack?

A Respiratory illness. There are some children who have a condition called exercise-induced bronchospasm where exercise and hyperventilation induced by the exercise trigger bronchospasm. I think those are two of the more common. . .

Q When you say "respiratory illness," what illnesses does that include?

A Any upper or lower respiratory illness.

Q Pneumonia?

A That would be an example of lower respiratory illness.

Q Bronchitis?

A Bronchitis, yeah.

Q Bronchialitis?

A Well, bronchialitis is distinct from asthma. In fact, it's one of the things that is difficult in the child less than two to differentiate from asthma.

Q Is otitis media a risk factor for asthma, for inducing the asthma attack?

A Not directly, no.

Q But again, it might be indirectly because of the relationship between that and the upper respiratory tract infection?

A Right. Exactly.

Q Is owning a furry pet a risk factor for inducing an asthma attack?

A If the individual is sensitive to the dander of the pet, the coat of the pet, yeah.

Q But it would be a risk factor on a population basis, wouldn't it?

A Exactly.

Q Any pets worse than others?

A Well, I think the most common one that are risk factors are dogs and cats, horses. Other animals that shed also increase the risk of exacerbating asthma.

Q Does the inadequate cleaning of surfaces

in a home, especially rugs -- is that a risk factor for inducing an asthma attack?

A Not really, no.

Q Is it statistically linked with the incidence of asthma attack?

A Rather than not cleaning them, cleaning them sometimes induces an attack of asthma. In other words, as long as the particulate matter is compacted in the floor covering, it's less of a risk than if it's up in the air and able to be inhaled.

Q So sometimes when you clean it, you stir things up and it becomes airborne and can be inhaled?

A Uh-huh.

Q Hasn't it also been reported, though, that inadequate cleaning itself has been linked with the induction of asthma?

A Not that, per se. I mean, there are other aspects of that that probably --

Q Is the failure to change air filters in a home a risk factor for inducing an asthmatic attack?

A It's a difficult question to answer. I mean, air filters, as long as they are functioning, would be protective against particulate matter getting into the air. If they are past the point of

where they can trap particles, some might get through, yeah.

Q Is there a -- I guess some of these questions are getting at perhaps it could be captured broadly as a basic hygiene issue.

A Uh-huh.

Q Is there a link between basic hygiene in the home and the induction of an asthmatic attack in a child?

A What do you mean by "hygiene"?

Q Again, cleanliness of the home, maintenance of the filters, cleaning the carpets.

A I think the overall aspect of whether a home is clean or dirty -- certainly, if there is a lot of particulate matter in the area, it's of greater impact on any kind of respiratory illness, yeah.

Q Is the use of a humidifier in a child's room a risk factor for an asthmatic attack?

A Yes, it can be.

Q Is the failure to breast-feed a child during its infancy a risk factor for subsequent frequency of asthmatic attacks?

A No.

Q Is exposure to cockroach antigens a risk

factor for asthmatic attacks in children?

A Exposure to cockroach antigens; how do you mean that?

Q I guess this would occur by virtue of the inhalation of particles from decaying cockroach carcasses in the home.

A I've never seen anything on cockroaches, but inhaling any particles would be a risk factor.

Q You've never seen anything linking cockroaches in the home with asthmatic attacks?

A No. I think the most popular one is the dust mite in that there is the best data on that.

Q What is the magnitude of the risk from exposure to dust mites for an asthmatic attack?

A It's significant.

Q Over 2?

A Well, I think when I say "it's significant," I think if you take a population of reactive airway-diseased children and put them in an environment in which there's no dust, they do much better. I think that's significant.

Q What do you believe to be the most important risk factors for an asthmatic attack?

A Most important?

Q The most important.

A Well, I think the major issues would be inhalant allergants.

Q Does the incidence of asthmatic attacks vary seasonally?

A Yes, they do.

Q Why is that?

A No one's quite certain about all of the variables. For example, in this state 50 percent of our cases occur in four months. And they're in the late spring or early summer and late summer/early fall. And the quotes are that it has to do with the prevailing winds, the warmth, the ability of inhalants to get into the air.

Q Would that include pollen in the air, for instance?

A Pollens, yeah.

Q Do you have any knowledge of the seasonal variation of asthmatic attacks in Mississippi?

A No.

Q Do you have an opinion as to what the impact of banning smoking today would be on the incidence of asthmatic attacks in Mississippi among the medicaid population?

A I have no idea of the magnitude.

Q I want to talk to you now about respiratory illnesses, including pneumonia and bronchitis.

Are those lower respiratory tract illnesses?

A Uh-huh.

Q Do you have an opinion as to the consequences of environmental tobacco smoke exposure for the incidence of lower respiratory tract illnesses?

A Are you talking about in infants or children or the newborn or. . .

Q Well, could you express your opinion based upon the age group, if that's what you need to do?

A Okay. I think, specifically, most of the respiratory illness in childhood that leads to significant compromise in the health status of the child exists between the ages of birth and about two years of age. That's the highest incidence of respiratory illness and account for over 60 percent of the admissions of children to hospitals.

And in terms of exposure to cigarette smoke and its impact, I think it's perhaps the most significant area where cigarette smoking impacts on

children's health that I'm familiar with.

Q Lower respiratory tract illnesses?

A Yes.

Q And can you describe the dose-response relationship for cigarette smoke in lower respiratory tract illnesses in children?

A Dose response. I don't think that anybody's put children -- I think it would be difficult to get them in terms of through any research committee. I don't think anybody's put children in an environmental chamber and measured the impact in terms of a dose response.

Most of it's been done by questionnaires of families and the documentation that the questionnaire's been accurate in -- some of the studies have been done on the basis of documenting cigarettes' exposure by measuring the metabolites of nicotine in the blood of the children, and there is a relationship in terms of the levels of cotinine, which is a metabolite of nicotine in blood levels.

Q Between those levels and?

A And the impact on a condition called wheezy bronchitis.

Q What is wheezy bronchitis?

A Well, under the age of two, there is --

again, the thought is that at least 60 percent of children experience an illness, a respiratory illness in which wheezing is a component.

In addition, the majority of those, over 60 percent, again, will not develop asthma. And these illnesses are due to the size of the airway being small and the fact that the data shows that children of smokers, even without a respiratory illness, have significantly greater reduction in airway size than children of the same age, same socioeconomic background whose parents did not -- mother did not smoke during pregnancy, so that even with upper respiratory infections being mild, these children are more likely to run into the complication of a wheezy bronchitis.

Q Are there any particular studies that you rely on for your opinion with respect to wheezy bronchitis?

A Yes. There is a number of studies coming out of the East Boston trials. The authors include Ira Tager, Scott Weiss, John Hanrahan. There's one by Brown, but John Hanrahan's work that they all have published together, but they take turns in being the senior author on the papers. But I think John Hanrahan's work is probably the most

careful.

There's another study published last week out of Australia by Peter Sly and his group showing essentially the same thing using different techniques but confirming the work done by Hanrahan in Boston.

Q And these are all studies of this wheezy bronchitis?

A No. They're of the size of the airways and the amount of flow limitation, that is, how fast the infant can empty his airways when he exhales. And they also -- the Boston group has also shown that these children, when they get illnesses, have a higher risk for wheezy bronchitis.

Q Are you able to attach an odds ratio or relative risk to parental smoking and the incidence of wheezy bronchitis?

A I'm not able to, but they -- because I just don't remember the numbers that have been applied, but these authors do -- have published on that.

Q Have any of these studies been conducted in a medicaid population?

A Yes.

Q Which of them have been?

A I think -- I don't know what percent of Hanrahan's patients offhand were on medicaid, but there's a significant percent.

Q Where was that study conducted?

A East Boston.

Q What are other risk factors for this wheezy bronchitis?

A Well, as I said, while 60 percent of these children never wheeze after the age of two, the other 40 percent will probably continue wheezing at intervals, and some of them will, in fact, have hereditary predisposition to asthma.

Q What's the medical treatment? Is there a medical treatment for wheezy bronchitis?

A Well, that's highly debatable. The problem with that is that it's a mixed group, some who have structural abnormality of the airway, that is, smaller airways; and some who have reactive bronchospasm, that is that their airways are twitchy, are more reactive, and respond to inflammation of the airways by going into spasm. So that the studies have been -- the outcome of studies using, for example, bronchodilators or anti-inflammatory agents have been conflicting because of the cause of the wheezing. In some swelling of the airway and smaller

airways, then bronchodilators' agents to dilate the airways is going to be ineffective. Where if this is a child who is really presenting early with reactive airways disease, bronchospasm, then bronchodilators can be highly effective.

So the problem with studies or many of the studies is that they haven't really accounted for the various variables such as, "Do your parents have asthma?" "Does another sibling in the family have asthma?" "Were you born prematurely?" Those kinds of questions.

Q So those studies may not actually have been looking at a homogeneous population. They may have had different underlying causes among the studied subjects?

A Right.

Q And efficacy of it varies depending on the underlying cause?

A Yes.

Q What other factors are statistically linked with the incidence of incidence of wheezy bronchitis?

I think you began to name a few a moments ago. Other siblings with asthma?

A Yes. I said family history of asthma,

whether it's parents or siblings; the incidence of early infancy, specially newborn; respiratory problems such as respiratory distress syndrome or prematurity, mecomium aspiration syndrome, other infectious causes that led to assisted ventilation in the newborn period; recognition of a condition called bronchopulmonary dysplasia.

There are a number of disorders which have a sequelae of more irritable airways and the propensity to wheeze with respiratory infections.

Q Are parental respiratory illnesses statistically linked with the incidence of wheezy bronchitis in infants?

A Are parental?

Q Parental.

A I would think they would be. I haven't seen any data, but it sounds reasonable.

Q Would that include parental history of bronchitis, for instance?

A No.

Q A parental history of asthma?

A Parental history of asthma, yes.

Q Parental history of emphysema?

A Not necessarily, no.

Q Would a history of wheeze in other

siblings be statistically linked with wheezy bronchitis in infants?

A If you're asking whether siblings' wheezing suggests a genetic predisposition to that, I would think I could answer affirmatively if ultimately the other sibling turned out to have reactive airways disease or asthma.

Q Is the use of kerosene stoves in a home statistically linked with wheezy bronchitis?

A I think it's linked with respiratory illness. That's true.

Q What about the use of gas stoves?

A I would think that any vapor that was able to -- any liquid that was able to be vaporized and is noxious to the airways would lead to wheezing or other kinds of respiratory embarrassment.

Q If a child comes in your office with wheezy bronchitis, is there anything about that child that would allow you to link it to exposure to cigarette smoke?

A I'll answer it the same way I answered the other; not unless I had a very strong history of cause and effect.

Q Nothing about the physical presentation of the child or any tests that you could perform on

the child?

A Not a readily available clinical test, no.

Q Do you have an opinion as to what the impact on the incidence of wheezy bronchitis in the Mississippi medicaid population would be if smoking were banned tomorrow?

A The magnitude, no.

Q What are the most important steps that could be taken to decrease the incidence of wheezy bronchitis in the Mississippi medicaid population?

A I think parental education. I think that's far and away -- when people look at the control of respiratory illness, knowledge about how respiratory illnesses occur and the steps to be taken to avoid it, I think education plays the most important role.

Q What factors should the parents be educated on?

A Well, just the steps to take to avoid respiratory illness. If you'd like, I'll give you an example.

Q Please.

A Well, first thing is better infection control when one member of the family has an illness,

such as hand-washing, which leads to protection against spreading respiratory illness.

It's usually hand transmission of the usual respiratory viruses rather than cough or inhalant particles from others. That would be one example of it.

Some things that we do in our clinic is talk about isolation to some extent from other children with respiratory illness when you can avoid it. We talk to our families about avoidance of cigarette smoke, either directly or indirectly, by, if somebody's got to smoke, that they change their clothes before handling a child who may be sensitive to it and certainly not to smoke in the child's presence.

There are climatary factors such as the in-house environment; avoidance of molds on windows, talking about getting the children out of the house for at least an hour or two after vacuuming the carpeting.

Those are all educational issues that can reduce the risk of problems with respiratory illness.

Q Thank you.

Doctor, in the 26(B)(4) Statement, it is

stated that you may express opinions about the relationship of impaired pulmonary function in sudden infant death syndrome.

A Yes.

Q What are your opinions in that regard?

A That there is body of data or literature that suggests that infants of mothers who smoke have an increased risk for abnormal respiratory control, one of which one of the syndromes of which is sudden infant death syndrome.

Q Did you review the literature with respect to the studies, the epidemiologic studies that have examined sudden infant death syndrome and parental smoking status?

A Uh-huh.

Q What did you find?

A That there's an association between parental smoking and the incidence of SIDS.

Q Did you also find studies that reported no association?

A I'm not sure I did. I might have.

Q You don't recall?

A I don't recall, no.

Q Did any of the studies that you reviewed present any data with respect to a dose-response

relationship between parental smoking and the incidence of SIDS?

A No.

Q Was SIDS -- in the studies that you reviewed, was SIDS linked to parental smoking as well as maternal smoking?

A I think the more impressive association was maternal smoking; that is, again, paternal smoking is only additive and of far less importance than maternal smoking.

Q This is a horrible subject to even contemplate, but what are the medical expenditures associated with a case of SIDS?

A They are actually very small.

Q Is there anything about a case of SIDS about the victim that would allow you to link an individual victim with exposure to tobacco smoke?

A No.

Q What is the overall incidence of SIDS in this country?

A It's about one per thousand live births or somewhat lower in certain populations.

Q So it varies across populations?

A Yes.

Q And can you describe that variation for

us.

A Variation? Well, at the present time it seems that the most important variation is whether -- one of the more important -- I shouldn't say the most -- variations has to do with the position with which the baby is put down to sleep.

Q Is sleeping in a prone position statistically linked with SIDS?

A Yes.

Q Does SIDS vary by sex?

A I think more boys are affected.

Q How is a diagnosis of SIDS made?

A Well, it's diagnosis by exclusion, that is, that it's a sudden demise of an infant usually somewhere between four to six weeks of age and three months of age for which no other cause can be determined.

Occasionally, supporting evidence can be that the infant may have sustained a life-threatening event, a preceding event where the child was found without respiratory rate and close to death and was successfully resuscitated.

Q In that sense is a diagnosis of SIDS essentially a catchall diagnosis for death that cannot be otherwise explained?

A It's a diagnosis by exclusion, that is that careful investigation has led to all other commonly expected problems and not so commonly expected problems being excluded.

Q What are the risk factors for SIDS?

A Risk factors? Well, prematurity is a major one. Small for gestation-age infants. There are infants of substance abusers who are at greater risk. Those are the major -- some of the major known risks.

Q Is SIDS statistically linked with parental socioeconomic status?

A I think there it's somewhat difficult to really carefully define that because some -- the question is raised versus socioeconomic status. For example, the highest incidence is in the American Indians. Is it something to do with being an American Indian, or is it something to do with parental behaviors or socioeconomic status?

Q This is always the problem, isn't it?

A Exactly.

Q Is SIDS statistically linked with maternal education level?

A Yes. It's been found to be another association.

Q Is SIDS statistically linked with maternal age?

A No. I don't think that's significant.

Q Is it linked with the degree to which prenatal care was utilized?

A I really can't comment on that. I'm not sure.

Q Is it linked with the marital status of the mother?

A I don't think so in that I think it would have to be linked to other causes. Marital status alone I don't think is a risk factor.

Q Does the incidence of SIDS vary seasonally?

A Yes.

Q Why is that?

A There are many things about SIDS which are yet to be defined. Some of the speculation -- it's higher in the winter, and some of the speculation has to do with prevalence of respiratory illness during wintertime.

Q Is the incidence of SIDS linked with the sharing of a bed with another sibling?

A I don't think so, no.

Q Is the incidence of SIDS linked with the

failure to be breast-fed?

A I've seen papers that suggest that relationship, and I'm not sure I remember whether it's significant or not.

Q What is the relative risk for SIDS in sleeping in a prone position?

A Actually, that's difficult to answer because, in essence, it's been -- it's different in different populations; that is, it's been most significantly seen to be in -- changing position has been associated with a much more dramatic reduction in the incidence of SIDS in Australia and in Europe than in the United States.

Q Why would that be?

A I'm unable to comment on that.

Q I take it that there's just an awful lot about SIDS that's not known yet?

A Yeah.

Q Would you have an opinion as to what the relative risk would be for parental smoking and the incidence of SIDS in the Mississippi medicaid population?

A No.

Q Would you have an opinion as to what the most important risk factors for the incidence of SIDS

would be in the Mississippi medicaid population?

A No.

MR. PATRICK: I'd like to take a couple of minutes' break here, if we could.

MR. FURR: Sure.

(Recess from 2:20 p.m. to 2:25 p.m.)

BY MR. FURR:

Q Doctor, you've provided us about an inch of documents today; is that correct?

A Yes.

Q And those documents consist of copies of published articles entirely; is that correct?

A Yes.

Q We'll mark those collectively as Platzker Exhibit F for identification and copy them and return the originals to you.

That is all the questions that I have today pending the matter that we discussed earlier with respect to the subject of production of additional documents and the opportunity to review the documents that you've provided today.

I thank you for your cooperativeness and responses today. I believe Mr. Minton has some questions.

///

EXAMINATION

BY MR. MINTON:

Q And I will try and be fairly brief.

A Good.

Q This is more for my own education and perhaps clearing up the record, but just in terms of how we've been using some terms, I interpreted and may have misinterpreted a difference that you seem to draw between dependent variables and the confounders; was I correct in that?

A Yes.

Q And are we to understand that dependent variables, you have deemed those that -- you have deemed those to be variables that are related in some way to the variables?

A Directly related.

Q -- directly related to the variables at issue and confounders secondarily related, at best, or maybe unrelated to the variables at issue?

A No. Other variables are variables that have less importance but still must be taken into account before the subject at hand can be resolved.

Q And when we have discussed socioeconomic factors as we have in the context of many of the health effects that have been discussed here, would

those typically -- in the scheme of classification that we have used, would those be better categorized as confounders as opposed to dependent variables?

A It would depend on the issue at hand.

Q So it's going to be context-specific in each case?

A Yes; exactly.

Q All right.

Another rather broad question; is it fair to say that it is not the intent of any of your opinions to comment on the health impact in the medicaid population in Mississippi of cigarette smoking with respect to any of the health outcomes or health effects that are listed on Exhibit B?

MR. PATRICK: I'm going to object to the form.

You can answer.

THE WITNESS: I would say quantitatively, the question is directed to me related to whether I can put a number on the increase in risk specifically to populations in Mississippi.

BY MR. MINTON:

Q And you have declined to do that?

A I have declined to do that.

Q All right.

And quantitatively with respect to any

dollars that may or may not have been expended with respect to illnesses alleged to be related to cigarette smoking, the same would be true; you have declined to attempt to put any sort of number on that?

A Yes.

Q In describing the project as it was presented to you originally -- I believe you said Ms. Flowers had been the initial contact -- you'd used the word "hypothesis" and the hypothesis that you were asked to examine, and that was is cigarette smoking related in some way to various health end points that relate to your particular clinical practice?

In terms of examining that hypothesis, have you done that as a clinician rather than a person attempting to review the epidemiologic literature and comment on medical causation as that term is used in the epidemiologic literature?

That was a pretty long-winded question. I don't know if you --

A Perhaps we should break it into parts.

Q Are you aware in terms of the experience that you have had with epidemiology that there are various criteria that are used to look at statistical

associations that have been discovered in particular epidemiologic studies, to then analyze whether or not those statistical associations are quote, unquote, causal associations?

Are you aware that's an epidemiologic constructor or that is an epidemiologic analysis that is performed that, once there has been a report of a statistical association, that there are then further criteria that are applied to determine whether or not that statistical association can be denominated a quote, unquote, causal association?

MR. PATRICK: Objection to form.

You can answer.

THE WITNESS: I think that's a difficult question to answer in that there are some studies in which one addresses a particular population and looks at whether there is an illness associated with that population, and it then corrects for the various variables and then looks again statistically.

And if at that time there seems to be an association, one will agree that, even when the epidemiology isn't fully understood, that one particular variable has an impact on the disease, and that would be fairly standard epidemiologic practice.

Some of the data that we've reviewed --

and that is why I'm being more specific -- an experiment has been done, that is, a particular population has been looked at. And where the end point wasn't an illness but a change in morphogenesis, that is, the development of the individual, various experiments have been done to link the two by looking at the dose of the tobacco smoke and then the impact on the morphologic end point.

And in those instances the huge population in that the same statistical analyses, while still active, are perhaps less important than the associations being made; that is, you've excluded various variables. Albeit the population is smaller, you have evidence of the dose response that you can be fairly certain is existent.

I don't know if I've answered your question, and I'm not totally certain I understand yours.

BY MR. MINTON:

Q Are you familiar, for instance, with the A.B. Hill criteria --

A No.

Q -- Brandford Hill's criteria in terms of taking a statistical association and examining

whether or not that statistical association can be called a "causal association"?

A No.

Q Was there some particular meaning that you were attempting to impart by using the word "syndrome" on Exhibit B?

A Syndrome in what context?

Q Fetal tobacco syndrome is the --

A Yes.

Q What was the meaning attached to the word "syndrome"?

A These are an associated series of findings that come together and on a statistical basis are thought to be a phenomena.

Q As for each of those phenomena, there are alternative causes that have been ascribed other than maternal cigarette smoking or ETS?

A For each one of the components, there are possibly other factors that have been related, but the overall impact of them coming together is an association that has been sustained by statistical analysis sufficient that the center for disease control in their publication "MMWT" has adopted the term.

Q The relative importance of the

alternative causes in terms of the relative risk will vary from population to population for each of these health end points, won't they, Dr. Platzker?

A Yes. It depends, as I said, on the ability of each population to handle the same stress, same stimulus.

Q And the relative importance of the other risk factors that were or were not present in each of those populations; correct?

A Well, I'm assuming that in any study we talk about we've accounted for those risk factors.

Q Because, as you said earlier, you cannot take the results from study A in population A and apply it to population B until -- unless and until you have controlled for each of the other --

A Exactly.

Q -- dependent variables and confounders?

A Correct.

Q As a matter of fact, proper scientific method would require that, before we take any results from study A and population A and attempt to apply it to population B, that we do just that; we control for each of those dependent variables and confounders?

A As many as you can.

Q And in terms of your analysis of the

literature in this case, Ms. Flowers never asked you to do that, did she?

MR. PATRICK: I'm going to object.

You can answer.

BY MR. MINTON:

Q Were you asked, "Dr. Platzker, I want you to do a rigorous analysis of these studies and attempt to see to what extent each of the populations in those studies resembled or did not resemble the Mississippi medicaid population in a manner that would allow you to control for those confounders and dependent variables"?

A No.

Q And unless and until that analysis was made, in other words, with respect to each of those studies, we determine what the confounders and what the dependent variables were and see how they matched with the Mississippi medicaid population, it would be inappropriate to take relative risk data from those studies and attempt to apply it to the Mississippi medicaid population; isn't that right?

MR. PATRICK: Objection.

THE WITNESS: I can't answer that question because it's too broad.

///

BY MR. MINTON:

Q Until we took data from the Mississippi medicaid population and then analyzed that data to see how it comported with the data from each of the studies that are identified in Exhibit B, we would be unable to determine the presence or absence of confounding and dependent variables and, therefore, the presence or absence of a suitable surrogate population from which to take data and apply it to the Mississippi medicaid population?

A I don't agree with that, no. I mean, there's certain things that have been clearly documented, where populations and their socioeconomic background and health status of the mother, drug abuse, whatever, have been taken into account, and still the difference has been confirmed as to the impact of smoking of cigarettes on the fetus.

I mean, the strongest data is the data coming out right now on airway caliber and restriction of expiratory flow in infants whose mothers smoke, and this is data that's now been confirmed in several populations and seems to be a biologic phenomenon that occurs. And that particular issue is fairly tight.

Other issues that have been rejected as

causal, cause and effect, with regard to cigarette smoking is much less well-established, and in those issues you might really have to go and look at the demographics of the population in Mississippi to sustain some hypothesis that those particular aspects have been wrapped up as associated with tobacco smoke exposure.

Q Was there a particular health effect, then, that you believed had been associated so strongly with cigarette smoking in the studies that you've identified here that you did not believe analysis of the Mississippi medicaid population for their demographics and their dependent confounding variables would be necessary in order to make some conclusion?

A Sure.

Q Which one was that?

A I would think the data, the strongest data, for example, an Apgar score, which is the evidence of current and preexisting hypoxemia, lack of adequate oxygen in the blood -- the data there, the only reliable data suggests that mothers have to smoke 31 to 40 cigarettes a day to significantly reduce the Apgar scores suggesting increased fetal hypoxemia.

Now, I don't know enough about the obstetric population, maternal population, in Mississippi to really comment on that, whether you -- you know, what the cigarette -- the dose versus the blood levels of the various components of cigarette smoke would be in the fetus and whether at 31 to 40 cigarettes a day you'd see a difference.

The only thing I know, for example, is that -- and I would suspect it's probably still true in Mississippi, that there's a higher incidence of a larger portion of the population being black, and I know that blacks metabolize cotinine, which is a by-product of nicotine, less well than white populations.

And I'm not certain whether you need 31 cigarettes a day smoked to produce that kind of impact on the Apgar score. It may or may not be true in Mississippi, and that particular bit of information I would want to see confirmed by some studies if I were to go out on a limb and extrapolate this one very important, albeit important study to the population in Mississippi.

Q But at this point without Mississippi data you would be scientifically quote, unquote, going out on a limb in order to make some comparison

between the data in the studies and the Mississippi medicaid population?

MR. PATRICK: Objection to form.

You can answer.

THE WITNESS: Well, I think that's -- as I said, it may clearly be. The question is whether you need to smoke a pack and a half of cigarettes a day to get that effect in Mississippi. Other populations that have been studied show that a pack and a half a day is sufficient to do it.

BY MR. MINTON:

Q Are you familiar with any of the studies which have shown no difference in Apgar scores based on maternal smoking or ETS exposure?

A Yes.

Q How did you weight them in terms of your analysis of the studies?

A Well, in each of those studies, they didn't use all of the mothers smoking more than -- I think it was ten cigarettes a day were lumped into a single group, so that those who smoked more may not have influenced the data enough, that is, the standard deviation -- that group may have been larger -- and that you would lose the top end of those consuming the most cigarettes.

Q Do you know how many subjects Garn had in the high-dose portion of his two studies?

A I can't remember.

Q Would it be significant to you whether that was a large group of people or a small group of people?

A I think so. It depends, I mean, statistically, how tight the data was. I can't offhand remember.

Q Other than Garn's two studies, are you aware of anybody else who's looked at that quote, unquote, high-dose group?

A No.

Q And if Garn studies less than a hundred people, would you think that that was sufficient in order to make some sort of --

A I'd have to look at the data to tell you.

Q There's a possibility that investigating less than a hundred people would be sufficient to make some broad-based generalization?

A Yes.

Q Other than Apgar scores, are there other studies that you think could be applied regardless of demographic differences between Mississippi and the

study population at issue?

A Yes.

Q Which would those be?

A Well, I've already made the statement that I think that the issue of post-natal smoking in terms of airway function, I'm fairly confident that data is going to stand up, whether it's Mississippi or Indiana or China.

I think the amount of data coming out from laboratories using different techniques to study the same phenomena and getting the same results strong enough that it's going to be hard to refute that data. That's a biologic phenomenon that's been reasonably well-documented.

Q What is the relative risk for post-natal dysfunction, airway dysfunction in parents who smoke -- in the children of parents who smoke?

A Infants who are delivered to mothers who have smoked during pregnancy in the East Boston study, Australian studies of Sly and Swift suggests that, even at low doses, there's a significant reduction in the capacity to exhale in infants.

And this, then, population when followed has a much higher incidence of wheezy bronchitis in the first two years of life.

There are also studies looking at these same group of children from 6 to about 13 years of age in respect to their airway function, and it's found to be dysfunctioning.

Q There was a listing of numerous other alternative causes for wheezy bronchitis --

A Uh-huh.

Q -- that you and Mr. Furr talked about.

In terms of the relative risk ascribable to cigarette smoking, what is your opinion of what the relative risk contribution is for cigarette smoking?

A I think that the risk from cigarette smoking is pretty great because of the fact that these studies show that there is a reduction in expired flow, maximum expired flow in all infants, children, adolescents who are exposed to cigarette smoke, either in-utero and post-natally or post-natally alone.

Q Can you quantify the relative risk number for us?

A I wouldn't be able to give you an odds ratio because the data was not expressed in the literature in all of the studies as odds ratio. It was expressed as variation from the mean.

Q Wouldn't we need an odds ratio or a relative risk calculation before we could determine the relative importance of that risk factor against others so that we could determine how elevated that relative risk is with respect to other relative risks?

A Which ones had you in mind?

Q For any of the ones that you ranked for us earlier in terms of this particular disease. For wheezy bronchitis, for instance.

A Well, in the East Boston study, they were able to account for the incidence of prematurity, the incidence of respiratory distress syndrome, the incidence of meconium aspiration syndrome.

Those are the major neonatal risks for subsequent respiratory illness. And even with that, they were unable to account for the variation from normal which they found in these individuals.

Q In your opinion, did they account for all of the risk factors that are present for wheezy bronchitis?

A I think they accounted for all the major ones. There are two kinds of studies -- actually, three kinds of studies; one looking at airway

function post -- in the immediate post-natal period. And with Hanrahan data, they looked at it two weeks -- within two weeks after birth.

In the Swift/Peter Sly data, they looked at it within 48 hours after birth because the question raised by the Hanrahan data was the quantization of fetal effects versus post-natal exposure to parents who smoke.

Q Did they control -- did they have cross-match controls that they compared the incidence, then, against?

A They looked at smokers versus nonsmokers.

Q But they did not develop any sort of relative risk or odds ratio from that?

A No.

Q Has anybody developed a relative risk or odds ratio for wheezy bronchitis?

A I think from the data obtained, it might be possible to do that. They didn't do it. I didn't do it.

Q So you don't have any sense of what the relative risk or odds ratio is for cigarette smoking in that particular --

A Phenomenon.

Q -- phenomenon?

A No.

Q Did we exhaust, in terms of Exhibit B, then, with the Apgar scores and the post-natal airways function, the health effects that you believe can be viewed independently of an analysis of the Mississippi medicaid population?

A I think what we've done is to discuss those areas that I feel that I'm most capable of discussing.

Q And just so everyone's clear what that statement means, you would not be prepared to discuss the possible incidence or prevalence of smoking-attributable health effects in the Mississippi medicaid population outside those two areas without doing an analysis of the demographics of the Mississippi medicaid population?

A No. That's not what I said. I said that I have an area of expertise, an area of expertise limited to respiratory illness and respiratory function. Some of the -- some other attributions are development disability, mental retardation, behavioral issues.

And that's certainly not my area of competence. And while I've listed the studies, I'm

really not prepared to comment on their validity. The various techniques and methodologies that we use -- I mean, I'm familiar with some of the studies that have been done, but to comment on whether somebody would have used a different technique to measure behavior or development or intellectual capabilities, that's outside my range of expertise.

Q So in terms of taking the studies that are listed on Exhibit B, with the exception of studies related to Apgar scores and post-natal airway dysfunction, it's not within the scope of what you're opining on to say whether a causal association has been demonstrated in the Mississippi medicaid population?

A What I'm commenting on is that there are certain things that to be -- to provide a high degree of confidence, there are certain things as to whether there's an association between cigarette exposure, whether intrauterine or post-natal, and that phenomena, it may be beneficial to also have studies in Mississippi.

There are other things where independent of what population, the correlations have been so good that it's unnecessary, really, to do studies in Mississippi.

Finally, that there are areas where I've read about the issues and am aware of the data but am not a professed expert in those areas and would prefer to leave it to someone else to decide whether other studies should be done to either confirm the relationship or to associate the relationship to Mississippi populations.

I mean, I don't want to overreach what I feel I know on a firsthand basis from training and experience.

Q It seems to me you just listed three categories of areas. The first category of areas -- the first area was health effects where it would be important to know -- in assessing a causal association, it would be helpful or necessary to know the demographics and the particulars of the Mississippi medicaid population.

A No. That was the last area I covered. The first area is areas where a biologic effect has been demonstrated beyond the need for performing any further studies independent of the populations.

Q In other words, where a mechanism has been demonstrated?

A Right; as perhaps not the only mechanism but an important mechanism in the evolution of a

particular problem.

Q But even where there is a plausible biological mechanism that supports a statistical association that's been demonstrated by an epidemiologic study, that doesn't rule out the potential that there could be confounders or dependent variables in the Mississippi population that are also responsible for the occurrence of those health effects in that population.

MR. PATRICK: Objection to form.

You can answer.

THE WITNESS: I've already stated that there are other causes, for example, of lung hypoplasia, but both in animal models, the Sanco studies being one; and in human populations, the Hanrahan data and the data from Doyle, we've shown that exposure, intrauterine exposure to cigarette smoke is sufficient to cause lung hypoplasia, decreased lung tissue.

Whether it be more or less in the Mississippi population, I can't really say, but we know that given the amount of exposure -- and it's fairly modest exposures -- in both of those populations, they've shown an effect. In both of those study groups, they've shown an effect, and it

would be hard to -- inasmuch as there's a high percentage of the population being black, and blacks clear cotinine, detoxify or metabolize nicotine less well, it would be hard to believe that the effect would be less in the populations in Mississippi.

BY MR. MINTON:

Q Well, would we have to know, for instance, what the other alternative causes are for that disease and how they vary with the demographics of the population in Mississippi in order to make that statement?

A I don't think so.

Q Are there other risk factors that are related, you know --

A To lung hypoplasia?

Q Yes.

A Certainly. Congenital diaphragmatic hernia, which occurs in one out of every 2,100 births, certainly would be. But it's a very rare, relatively rare occurrence.

There are chest wall deformities such as would occur in some kinds of dwarfism, but that's also fairly rare in occurrence, so that the known forms of lung hypoplasia are fairly rare.

However, the exposure to smoke is fairly

frequent. And in animal populations where it's been studied, either in rats or in fetal lambs, it's been shown with fairly modest -- and you'll have reprints of those articles -- modest exposures that it is cause and effect, not to say that it's the only cause. But the other causes in humans are fairly rare in terms of lung hypoplasia.

Q Lung hypoplasia, for instance, isn't associated with prematurity?

A No.

Q Or any of the other more common things we've been talking about?

A No.

Q Is lung hypoplasia the only category of health effect that you do not think is related to demographic factors in any way? That's listed on Exhibit B.

A I really think that I haven't reviewed each one specifically, but I think it would depend on the populations being studied.

For example, there are a host of studies. And, in fact, there's even a meta-analysis that has been performed on the issue of intrauterine growth retardation or small for gestation age, and the great preponderance of the studies and the

meta-analysis confirmed that mothers who smoke -- after taking into account and controlling for the other variables, mothers who smoke have smaller babies.

And again, I mean, I think the number of studies that have been done and coming at it from many different directions really don't -- really point out that further studies, regardless of the population, are unnecessary.

I mean, there are other findings, obviously, that -- and I pointed out the one with low Apgar scores -- that I think it bears some scrutiny, and really the last, I'd like to see more studies of that issue in well-controlled populations to really be able to make a decision on dose response and whether it's a significant change variation from normal; whether the confidence limits are tight enough, so that I think it varies on a case-by-case basis.

Q Are you suggesting, Dr. Platzker, that there has been a study done, for instance, of intrauterine growth retardation that has controlled perfectly for the differences between smoking and other socioeconomic or demographic differences that may be associated with smoking?

A I don't think that there is any perfect study that's been done. That's probably why we continue to this day to see more studies done trying to more tightly control for the many, many variables.

But all of the really outstanding studies come up with same conclusions. Some of the studies, in fact -- and I go back to the national collaborative study in the late '50s -- tried to control for so many variables that they were unable, really, to make a great deal of sense out of some of the questions that they asked, so that there is no -- we know there are very few perfect research studies in the human populations because we have difficulty in controlling for every variable.

Q Did you identify in this list, for instance, of the intrauterine growth retardation studies, that you thought had done a particularly good job in controlling for variables?

A One study that -- the Martinez, et al., in a 1994 study, looked at the Tucson population. There's a long-term health outcome study, and Fernando Martinez was part of the group. And this is a study that looked at adults and the evolution of the decline in lung function in adults and first associated more rapid decline in lung function with

the childhood respiratory troubles, as well as the use of cigarette smoking.

And he looked at the population at the other end of the spectrum looking at what the infants looked like at birth from mothers who smoked. His area is, again, respiratory control but as a by-product -- respiratory function, but as a by-product of that, they had a publication on weight, and that was a fairly nice study.

Again, it wasn't a perfect study because it's rather difficult and costly to control for all of the variables.

Q And in terms of weighting the variables that are involved, are the clinical risk predictors that we've talked about, like the Creasy Scale, are those the ones that clinicians would look to in terms of the relative weight of those risk factors?

A You know, in the review I did, very few -- none of them, in fact, talked about risk -- the Creasy Scale. And in fact, there was one paper here in 1993 that Castro Azin studied. Azin's a statistician -- had one of the really famed perinatologists, Calvin Hobo, involved in the study. They didn't mention the Creasy Scale at all -- I'm surprised -- or at least I don't remember them

bringing it up.

Q As you sit here today, do you know what the relative weights are from any scale among the various risk factors for intrauterine growth retardation?

A I think people have assigned variable risks, and it's -- whether they've controlled for all of them in every study, I can't really say. I don't believe anyone has.

Q And truly that's the major impediment from taking any relative risk estimation and taking it from one population and attempting to apply it to another, isn't it?

MR. PATRICK: Objection.

THE WITNESS: Well, you know, I think you tend to place a tremendous value in relative risk. I place equal value in being able to look at populations as opposed to a mean and standard deviation curve. In a particular population exposed to a particular variable, all of the weight is that they fall below the mean and, in fact, have a disproportionate number below one and two standard deviations. I leave it to others to assess risk ratio and odds ratio to the currents in a specific individual.

See, the problem with odds ratio, it talks about an individual and not a population. For medical -- for physicians the importance is to know whether the risk is there from a population point of view because we counsel our patients on that basis rather than trying to look at an odds ratio.

In other words, prevention looks at the potential that someone will be in a high-risk group and helps the patient move from that high-risk group outside of it.

BY MR. MINTON:

Q Is that how you reviewed these studies --

A Yes.

Q -- from that perspective?

A Right.

Q So from your perspective, a study that maximized sensitivity because it would pose the least chance of including -- excuse me -- because it posed the smallest chance of a false negative, that that was the perspective that you brought to the analysis of these studies?

MR. PATRICK: Objection to form.

You can answer.

THE WITNESS: I don't understand exactly what

you're stating.

BY MR. MINTON:

Q Would sensitivity normally be defined as the likelihood that, if a person has the disease, that they will test positive under whatever the risk rating methodology is used?

MR. PATRICK: Same objection.

THE WITNESS: If what you're saying is, if I'm exposed to streptococcal disease, the likelihood is that I will develop streptococcal infection, and my advice to a patient so exposed is not to be exposed, then you're right; if there is a likelihood of an association, you want to avoid the association.

The associations depend on fairly good diagnostic skills, that is, the throat culture of the infected person being obtained before advising the patient to avoid them.

I don't know -- there are two issues; one is specificity; that is, is X related to Y?

Two, the sensitivity in the test that you do to relate X to Y, is it so sensitive that it's going to pick up scatter and not really an association, or is it totally insensitive and, when the association is present, cannot pick it up? We try to err on the aside of being -- using tests that

are highly specific and sensitive. That is, at least, my opinion.

BY MR. MINTON:

Q Dr. Platzker, has there been any consistent pathology described with respect to infants who have died of sudden infant death syndrome?

A Consistent pathology with regard to what organism?

A Any organism.

Q Well, most -- the difficulty is that we're seeing a dead -- somebody who died, and pulmonary edema has been commonly associated with it. More recent data suggests that they have more numerous cells, called "neuroepithelial bodies," in their respiratory tract, but is there isn't a totally classic pathology that makes it distinct from any other disease.

Q Are you familiar with Brad Thatcher's work at Washington University Children's Hospital with SIDS?

A Yeah. We're close friends.

Q And what scientifical validity do you find in his determination about the carbon-monoxide-retaining properties of pillows and

blankets being closely associated with SIDS?

A I think he did some very valuable studies in terms of looking at respiratory control in infants who were asleep.

I think that particular study that you're quoting is a nice contribution, but it's -- in terms of the overall impact on the disease and its causation, I think other than the sleeping position and the risk of using fluffy pillows, there isn't too much that I can draw from that study.

I think it's -- the result is more or less expected.

Q Have you completed all the work that you've been asked to do in this case?

A I was just asked to review the data and be available for deposition.

Q Was there any specific data that you were asked to review, or was that left up to you entirely?

A That was left totally up to me; no instruction.

MR. MINTON: Off the record.

MR. FURR: Off the record.

(Discussion held off the record.)

(Recess from 3:20 p.m. to 3:25 p.m.)

BY MR. MINTON:

Q Dr. Platzker, with respect to intrauterine growth retardation, are there medical expenses that are consistently clinically related with intrauterine growth retardation?

A The majority of babies with intrauterine growth retardation are not acutely ill. There are, however, other infants who suffer from the lack of energy stores during delivery, and those infants are at high risk at birth for having a condition called "hypoglycemia," for having hypocalcemia, for having low Apgar scores. And if that isn't treated rapidly, for having neurologic dysfunction. As you know, hypoglycemia can cause seizures.

These infants also may experience hypertension and have increased metabolism because of the fact that thyroid function seems to be more mature in these infants.

Q Are those because of morbidity that tends to be associated with IUGR, or are those all possible sequelae of IUGR?

A No. I think it's part and parcel of IUGR. It's a fairly well-known syndrome in which one of the mechanisms for it is that there's an inadequate placental circulation, and the amount of

nutriment that the fetus gets is less. The fetus then tries to use it more efficiently. And in many of the instances of intrauterine growth retardation, weight is sacrificed, in kind, to preserve nutrition to the brain so that weight is reduced, but length and head circumference are less reduced.

When these infants come to the point of delivery, they have less glucose stored in the heart and the liver and in the muscle such that they tend to consume these stores and in a usual birthing process, rather than having four-to-six hours' worth of glucose stores, these infants may already be suffering from lack of glucose for energy stores.

This forces the baby to consume its fat and protein, and the by-products of both of these are acids, and these babies at birth are acidotic, and, in essence, are very much like diabetics in ketoacidosis. They breathe rapidly. They excrete more acid in their urine trying to get rid of acid. They have low blood glucose rather than high blood glucose, and they require fairly immediate care to get them over this situation.

Q What percentage of babies with intrauterine growth retardation have no associated clinical problems?

A I would think while they -- it's difficult to say because mild hypoglycemia -- you may not even notice it, but I think most of them -- if the obstetric care is reasonably good, most of them you would probably notice it or the amount of increased respiratory rate, heart rate, et cetera, may be missed thinking it's just the baby who's been delivered fretful.

So I think the majority probably do reasonably well from a layman's perspective.

Q And in terms of doing reasonably well, that means that they wouldn't have associated clinical problems that would require additional medical treatment?

A Yes. Uh-huh.

Q And in terms of those instances that constitute less than the majority of intrauterine growth retardation, can you give us the percentage incidence of --

A Untoward events?

Q Yes.

A 9 percent, 9.0.

Q So in cases where intrauterine growth retardation -- in 9 percent of those cases, there's some further medical intervention that occurs?

A I'm underestimating the -- I want to be conservative, but I know that in every study that's been done, 9 percent of babies are meconium state, that is, that they pass stool during the process of either labor and delivery, and this is evidence of distress such as they might experience with hypoglycemia or other factors associated with lack of oxygen.

And in those instances, those babies all require medical care at birth, and 20 percent of them will become quite ill requiring -- usually requiring assisted ventilation.

Q So 91 percent of --

A 91 percent --

Q 91 percent of IUGR babies are not going to require any additional medical intervention? 9 percent are going to require additional medical --

A At least 9.

Q -- at least 9 percent, and of that 9 percent, 20 percent, so about one point --

A 2 1/2 percent.

Q 2 percent are going to require a significant amount of medical intervention?

A Right.

Q Did we -- well, just to make sure that

we have, because we've reached a consensus that we don't recall whether we have, have we mentioned -- or let us go through, then, the other causes of intrauterine growth retardation.

A Uh-huh.

Q What other causes of intrauterine growth retardation are you aware of?

A Okay. The most common are severe malnutrition in the mother. I mean, I'm talking internationally. In Africa there is a very high frequency of severe malnutrition in the mothers, and this causes perhaps the most severe expression of intrauterine growth retardation.

In the United States this would have to do with class -- well, I won't get into classes, but the more severe diabetic in terms of diabetics will have infants who have growth retardation.

There are elderly mothers that -- usually, mothers, I think, over 40 years of age whose -- in which placentation, that is, the placenta becomes senile very early, lack of nutrition to the fetus, they will have infants who are growth-retarded.

Women who have very, very frequent pregnancies coupled with very poor nutrition tend to

have a greater risk for having infants who are growth-retarded.

There are various medical syndromes, albeit rare, which produce infants who are small for gestational age. These are often chromosomal disorders.

I might have missed one or two, but I think that's it.

Q Small stature of the mother, is that associated with IUGR?

A No. That generally isn't. I mean, small mothers have small babies so that --

Q It would be a secondary factor?

A It would be a secondary factor.

Q How about cardiac or pulmonary disease?

A Cardiac or pulmonary -- what do you mean?

Q Maternal cardiac or pulmonary disease.

A Any disorder which deprived the infant of adequate oxygen.

Q Anemia, for instance, maternal anemia?

A Severe maternal anemia might produce that.

Q Anything that would produce vascular compromise in the mother might be a risk factor for

IUGR?

A Like what?

Q Toxemia.

A Oh, I forgot. That's a very important one. Toxemia, very frequently, and severe toxemia has an association.

Q Alcohol use?

A Actually not, unless the use of alcohol is sufficient to cause the syndrome of fetal alcohol syndrome where there is clear expression of the morphologic findings.

Q How about stressful life events?

A Pardon me?

Q Stress as a confounder or as a dependent variable for IUGR?

A I haven't seen that described.

Q And in terms of relative risk, have you seen any data on relative risk of IUGR?

A No.

Q And is your opinion on IUGR like the other opinions that you've rendered herein with respect to, as far as we can determine, smoking is not a necessary or a sufficient cause of IUGR?

MR. PATRICK: Objection.

THE WITNESS: No. I think smoking is one of

the many causes of intrauterine growth retardation.

BY MR. MINTON:

Q In terms of estimating the risk of smoking against those other risks, is it possible for you to do it based on the data that you've reviewed?

A I think every study in which they've looked at smoking with regard to intrauterine growth retardation and controlled for other variables, they've still been able to come up with a graded dose response.

Q The quantification of the risk or the elevation in that risk is what?

A Just as I pointed out, the relative risk is, under ten cigarettes a day, there's usually a reduction in birthweight of about a hundred-and-eighty grams. It's almost double that over ten cigarettes a day, the risk ratio. I only remember one paper offhand that's about one point -- I can't remember if it was 1.65 or 1.85 odds ratio if one to ten cigarettes; and it's over 2.65 or something like that with 10 to 20 cigarettes a day.

I haven't seen -- many of the papers don't look at odds ratio. They tend to look at mean weights compared to populations that don't smoke.

Q Have you, in the context of the work

that you've done here, Dr. Platzker, attempted to determine whether there was an association between maternal cigarette smoking or ETS and the very low-birthweight babies?

A No, I haven't seen anything like that.

Q Just so we have the same sort of information about intrauterine growth retardation that we have about the other areas that have been discussed, would it be fair to say that you have no opinion on how a cessation of cigarette smoking in the Mississippi medicaid population would affect the incidence of intrauterine growth retardation?

A Quantitatively, no.

MR. MINTON: I think those are all the questions I have, Dr. Platzker.

MR. FURR: Okay. I guess we're done. What we need to do is to attach as the last exhibit this file of medical articles. He's got his originals back.

MR. PATRICK: We need to verify these are all here.

He's gone through them, but he can do that again.

THE WITNESS: This is actually more than just the articles. There is an expert in this area who I haven't contacted, but if you want to contact him,

the telephone number is there. I think we got everything.

(Defendant's Exhibit F was marked for identification and is attached hereto.)

MR. MINTON: Thank you, Dr. Platzker.

MR. FURR: Thank you, Dr. Platzker.

(Discussion held off the record.)

MR. MINTON: Dr. Platzker is going to read and sign the deposition, and we've all stipulated that it's not necessary for him to go to the court reporter's office to do that, but that she may send him a copy of the transcript so that he can check it over for accuracy and make any corrections that are necessary to make sure that things are accurately stated.

How many days do you need for signature?

THE WITNESS: If I'm in town, it's one thing. I can't really --

MR. MINTON: 30 days?

MR. FURR: 30 days upon receipt of the transcript.

(At 3:45 p.m. Volume I of the deposition of ARNOLD PLATZKER, M.D., was continued sine die.)

I declare under penalty of perjury that the foregoing testimony is true and correct.

Executed at _____,

this _____ day of _____,

19____.

_____

SIGNATURE OF WITNESS

STATE OF CALIFORNIA)
)
COUNTY OF LOS ANGELES)

I, Johanna C. Blankinship, CSR No. 8734, a Certified Shorthand Reporter in and for the State of California, do hereby certify:

That the foregoing deposition of ARNOLD PLATZKER, M.D., VOLUME I, was taken before me pursuant to Notice at the time and place therein set forth, at which time the witness was put under oath by me;

That the testimony of the witness and all objections made at the time of the examination were recorded stenographically by me and were thereafter transcribed under my direction;

That the foregoing is a true record of the testimony and of all objections made at the time of the examination.

In witness whereof, I have subscribed my name this 6th day of November, 1996.

Johanna C. Blankenship

Certified Shorthand Reporter
No. 8734